Massachusetts Institute of Technology
Department of Economics
Working Paper Series

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Working Paper 04-08

August 2003

Room E52-251
50 Memorial Drive
Cambridge, MA 02142

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* We thank David Card, Doug Almond, Josh Angrist, Gary Becker, David Cutler, John DiNardo, Carlos Dobkin, Mark Duggan, Vernon Henderson, Charles Kolstad, Jeff Milyo, Steve Pischke, and numerous seminar participants for helpful comments. Justin McCrary, Doug Almond, Fu-Wing Lau, and Anand Dash provided outstanding research assistance. Funding from NSF Grant No. SBR-9730212 and NIH (NICHD) Grants No. R01 HD42176-01 and R03 HD38302-01 is gratefully acknowledged.

# Air Quality, Infant Mortality, and the Clean Air Act of 1970


Abstract

We examine the effects of total suspended particulates (TSPs) air pollution on infant health using the air quality improvements induced by the 1970 Clean Air Act Amendments (CAAA). This legislation imposed strict regulations on industrial polluters in "nonattainment" counties with TSPs concentrations exceeding the federal ceiling. We use nonattainment status as an instrumental variable for TSPs changes to estimate their impact on infant mortality changes in the first year that the 1970 CAAA was in force.

TSPs nonattainment status is associated with sharp reductions in both TSPs pollution and infant mortality from 1971 to 1972. The greater reductions in nonattainment counties near the federal ceiling relative to the "attainment" counties narrowly below the ceiling suggest that the regulations are the cause. We estimate that a one percent decline in TSPs results in a 0.5 percent decline in the infant mortality rate. Most of these effects are driven by a reduction in deaths occurring within one month of birth, suggesting that fetal exposure is a potential biological pathway. The results imply that roughly 1,300 fewer infants died in 1972 than would have in the absence of the Clean Air Act.



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## Introduction

The 1970 passage of the Clean Air Act Amendments (CAAA) and the establishment of the Environmental Protection Agency (EPA) marked an unprecedented attempt by the U.S. government to mandate lower levels of air pollution. The stated goal of these efforts was to achieve air quality standards that "protect the public health." In the case of total suspended particulates (TSPs), widely thought to be the most pernicious form of air pollution, the EPA set maximum allowable concentrations that every county was required to meet.[1] This study uses the reductions in TSPs concentrations induced by the 1970 CAAA to estimate the effects of TSPs on infant mortality.

Figure 1 shows trends in average TSPs pollution and infant mortality rates for the entire United States from 1970-1990.[2] It is apparent that air quality improved dramatically in the 1970s and 1980s, with particulate concentrations falling from an average of 95 micrograms-per-cubic meter ($\mu g/m^3$) to about 60 $\mu g/m^3$. Almost all of the reduction occurred in two punctuated periods, 1971-1973 and 1980-1982. The figure also shows the large decline in the infant mortality rate over the entire period. Although both TSPs pollution and infant mortality rates trended downward, their time-series correspondence does not provide conclusive evidence of a causal relationship. First, the punctuated declines in TSPs are not mirrored in the infant mortality rate series. Further, other potential determinants of infant mortality (e.g., social insurance programs, medical technology) were also changing during this period.

Chay and Greenstone (2003) establish that most of the 1980-82 decline in TSPs was attributable to the differential impacts of the 1981-82 recession across counties. The study uses the substantial differences in air pollution reductions across sites to estimate the impact of TSPs on infant mortality. We find that a one percent reduction in TSPs results in a 0.35 percent decline in the infant mortality rate at the county level. Most of these effects are driven by a decline in deaths occurring within one month of birth,

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[1] In this period, the EPA defined TSPs to include all particles with diameters less than or equal to 100 micrometers ($\mu m$). The focus of federal regulation shifted to particulates with diameters less than or equal to 10 $\mu m$ ($PM_{10}$) and 2.5 $\mu m$ ($PM_{2.5}$) in 1987 and 1997, respectively.

[2] The TSPs pollution series is based on averages across the 1,000-1,300 counties a year with TSPs monitors, while infant mortality rates are derived from the entire U.S. and are for all causes of death. The counties with TSPs data account for upwards of 80% of the U.S. population. Unless otherwise noted, infant mortality refers to death within the first year after birth.

suggesting that fetal exposure during pregnancy is a potential pathophysiologic mechanism. Overall, the estimates imply that about 2,500 fewer infants died from 1980-82 than would have in the absence of the dramatic reductions in particulates pollution.

In view of the significance of a potential causal link between TSPs pollution and infant mortality, we believe it is important to validate the results from our analysis of the early 1980s. In this paper, we examine data on infant mortality and air pollution from the other major episode of substantial changes in TSPs levels in the early 1970s. We begin by demonstrating that these changes are largely attributable to the 1970 CAAA. This legislation divided all U.S. counties into TSPs nonattainment and attainment categories based on whether their ambient TSPs concentrations were above or below the legislated maximum. Industrial emitters of TSPs were subject to significantly stricter regulations in nonattainment counties than in attainment ones. We find that the entire decline in TSPs during the early 1970s occurred in nonattainment counties and that two-thirds of the 1971-1974 decline in these counties occurred between 1971 and 1972, the first year that the 1970 CAAA was in force. Consequently, this study uses nonattainment status as an instrumental variable for 1971-1972 changes in TSPs to estimate their impact on changes in infant mortality.

This research design has several attractive features. First, it is plausible that federally-mandated regulatory pressure is orthogonal to county-level changes in infant mortality rates, except through its impact on air pollution – thus, nonattainment status may be a valid instrument. Second, nonattainment status is a discrete function of the previous year's TSPs levels. This discontinuity in the assignment of regulations can be used to gauge the credibility of the research design. Third, our analysis provides direct and easily interpreted estimates of the air quality and health benefits of the 1970 Act – benefits that can be compared with the costs to industry of complying with the regulations.

Finally, this study provides a unique opportunity to cross-validate the results from our earlier work by examining a different design applied to a different era. In contrast to the recession-driven TSPs reductions of the early 1980s, we use regulation-induced changes that occurred during an economic expansion (1971-1972). Thus, any potential biases due to economic shocks are likely to be mitigated. Also, particulates pollution levels were much higher in the early 1970s than in the early 1980s. As a

result, we can examine a different range of the pollution-infant mortality function and analyze the health benefits at the federally mandated concentration ceiling.

We implement the design using the most detailed and comprehensive data available on infant births, deaths, and TSPs levels for the 1969-1974 period. We find that the federally imposed county-level regulations are associated with large reductions in both the TSPs pollution levels and infant mortality rates of nonattainment counties. We estimate that a one percent reduction in TSPs results in a 0.5 percent decline in the infant mortality rate at the county level. As in our earlier study, most of these effects are driven by a reduction in the number of deaths occurring within one month of birth, suggesting that fetal exposure is a potential biological pathway.

Our attempts to probe the robustness of these conclusions suggest that the instrumental variables estimates of the effect of TSPs pollution on infant mortality are far less sensitive to specification than conventional cross-sectional and fixed effects estimates. Further, the timing and location of the TSPs and infant mortality improvements correspond remarkably well. As a test of internal validity, we find that the regulation-induced TSPs reductions are uncorrelated with infant deaths due to accidents and homicides. Finally, nonattainment and attainment counties near the federal ceiling exhibit discrete differences in TSPs and infant mortality changes, but do not exhibit differences in other observable characteristics. This provides our most convincing evidence that the regulations are causally related to declines in both TSPs and infant death.

Overall, our findings support the conclusions that air pollution has a causal effect on infant mortality and that the 1970 CAAA provided significant health benefits by reducing TSPs concentrations. The estimates imply that roughly 1,300 fewer infants died in 1972 than would have in the absence of the Clean Air Act. Also, the results correspond well with the findings in Chay and Greenstone (2003).

The paper proceeds as follows. Section I provides background on the 1970 Clean Air Act. Sections II and III discuss the research design and the econometric methods used to implement this design. Section IV summarizes the data, and Section V presents the empirical findings. Section VI provides estimates of the economic benefits of the Clean Air Act regulations, and Section VII concludes.

## I. Background on the 1970 Clean Air Act Amendments

Before 1970 the federal government did not play a significant role in the regulation of air pollution, a responsibility left primarily to state governments.[3] In the absence of federal legislation, few states acted to impose strict regulations on polluters within their jurisdictions. Concerned with the detrimental health effects of persistently high concentrations of suspended particulates pollution, and of other air pollutants, Congress passed the Clean Air Act Amendments of 1970.

The centerpiece of the 1970 CAAA was the establishment of separate federal standards, known as the National Ambient Air Quality Standards (NAAQS), for six pollutants. The CAAA's goal was to reduce local air pollution concentrations so that all U.S. counties would be in compliance with the NAAQS by 1975 (with the possibility of an extension to 1977). The first step in this process was the assignment of pollution-specific "attainment-nonattainment" designations to all U.S. counties for each of the regulated pollutants.[4] For TSPs pollution the EPA was required to designate a county as nonattainment if its TSPs concentrations exceeded either of two thresholds: 1) the annual geometric mean concentration exceeded 75 $\mu g/m^3$, or 2) the second highest daily concentration exceeded 260 $\mu g/m^3$. This standard prevailed from 1971 until 1987, when the EPA shifted its focus to the regulation of finer particles.

To achieve these standards, the fifty states were required to formulate and enforce State Implementation Plans (SIPs) that specified precise abatement activities. For nonattainment counties, the SIPs detailed plant-specific regulations for every major source of pollution. These local rules ordered that any substantial investment by a new or existing plant must be accompanied by the installation of state-of-the-art pollution abatement equipment and strict emissions ceilings. The SIPs also set emission limits for existing plants in nonattainment counties.

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[3] Lave and Omenn (1981) and Liroff (1986) provide more details on the CAAAs. In addition, see Greenstone (2002) and Chay and Greenstone (2000).

[4] It is unclear whether the EPA assigned nonattainment status at the county level or at a more aggregate geographic unit in the early 1970s. Our analysis assumes that the assignment was at the county level. The rationale for this choice is discussed in the "Nonattainment Data" subsection of the Data Appendix. Further, below we find that using nonattainment designations at a more aggregate geographic level does not change this paper's qualitative findings.

In stark contrast to the oversight in nonattainment counties, the restrictions on industrial polluters in attainment counties were considerably less stringent. Large-scale investments, such as new plants and large expansions at existing plants, required the installation of less expensive (and less effective) pollution abatement equipment. Moreover, existing plants and smaller investments were essentially unregulated.

Both the states and the federal EPA were given substantial enforcement powers to ensure that the goals of the CAAA were met. To limit variation in the intensity of regulation across states, the federal EPA had to approve all state regulation programs. On the compliance side, states initiated their own inspection programs and frequently fined non-compliers. Further, the EPA was required to impose its own procedures for attaining compliance in states that failed to adequately enforce the standards.

The 1970 CAAA was signed into law by Richard Nixon on December 31, 1970. Four months later on April 30, 1971, the EPA announced the final publication of the NAAQS that specified the national standards for TSPs concentrations. On August 14, 1971, the EPA published "Requirements for Preparation, Adoption and Submittal of Implementations Plans" in the Code of Federal Regulations, which set forth how states were to write their SIPs in order to achieve compliance with the NAAQS by 1975. Finally, the SIPs were due to the EPA by January of 1972, the first year in which the CAAA was enforced. Appendix Table 1 shows the timeline of the key dates associated with the 1970 CAAA, and the Data Appendix gives additional details.

Henderson (1996) provides evidence that the regulations were successfully enforced. He finds that ozone concentrations declined more in counties that were nonattainment for ozone than in attainment counties. Chay and Greenstone (2000) find that TSPs levels fell substantially more in counties that were nonattainment for TSPs than in attainment counties after the passage of the 1970 CAAA and throughout the 1970s.[5]

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[5] Greenstone (2002) provides further evidence on the effectiveness of the regulations. He finds that nonattainment status is associated with modest reductions in the employment, investment, and shipments of polluting manufacturers. Interestingly, the regulation of TSPs has little association with changes in employment. Instead, the overall employment declines are driven mostly by the regulation of other air pollutants.

## II. Research Design

Previous research has documented a statistical association between TSPs concentrations and adult mortality.[6] However, the reliability of the evidence has been seriously questioned for several reasons. First, since air pollution is not randomly assigned across locations, previous cross-sectional studies may not be adequately controlling for a number of potential confounding determinants of adult mortality (Pope and Dockery 1996; Fumento 1997). For example, areas with higher pollution levels also tend to have higher population densities, different economic conditions, and higher crime rates, all of which could impact adult health. Second, the lifetime exposure of adults to air pollution is unknown. Many studies implicitly assume that the current pollution concentration observed at a site accurately measures each resident's lifetime exposure. Third, the excess adult deaths that are attributed to temporarily elevated air pollution levels in time-series studies may be occurring among the already sick and represent little loss in life expectancy (Spix, et al. 1994; Lipfert and Wyzga 1995).

In the absence of randomized clinical trials, this study uses the air quality improvements induced by the 1970 Clean Air Act Amendments in the first year that they were in force to estimate the impact of TSPs on infant mortality. A research design based on comparisons between nonattainment and attainment counties has several attractive features.[7] First, TSPs nonattainment status is associated with sharp differences in changes in TSPs across sites in the early 1970s. Panel A of Figure 2 shows the trends in TSPs levels from 1969 to 1974 separately for counties that were nonattainment and attainment for TSPs in 1972 – the first year of CAAA enforcement.[8]

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[6] Studies of the adult mortality effects of particulates pollution include Lave and Seskin (1977), Pope and Dockery (1996), Dockery and Pope (1996), Dockery, et al. (1993), and Pope, et al. (1995). Chay, Dobkin, and Greenstone (2003) examine the association between adult and elderly mortality and regulation-induced TSPs declines.

[7] Despite extensive efforts, we were unable to obtain a list of the counties that were initially designated TSPs nonattainment by the EPA. Given the timeline for states to comply with the CAAA, states likely ascertained the identity of the counties with TSPs concentrations above the NAAQS while writing their SIPs during the second half of 1971. We assume that they relied on the available TSPs concentrations data from 1970 to determine which counties would be nonattainment for 1972. Thus, we assign counties with TSPs concentrations exceeding the NAAQS in 1970 to the 1972 TSPs nonattainment category. All other counties with nonmissing TSPs data are designated attainment for that year. The measures of TSPs concentrations are derived from the same network of TSPs monitors used by the states and the EPA in this period. The Data Appendix provides further details on our assignment rule.

[8] The sample consists of the 401 counties with continuous monitor readings from 1969-1974 – 229 of these counties are TSPs nonattainment in 1972. Together these counties account for almost 60 percent of all births in the U.S. in

The figure documents that before the 1970 CAAA, TSPs concentrations were 40-$\mu g/m^3$ higher in nonattainment counties. While the pollution trends are similar in the two groups from 1969 to 1971, there is a sharp break in trend in nonattainment counties after implementation of the CAAA. From 1971-74 newly regulated counties had a stunning 20-$\mu g/m^3$ reduction in TSPs, while TSPs fell by only 3 $\mu g/m^3$ in attainment counties. These comparisons suggest that virtually the entire national decline in TSPs from 1971-74 in Figure 1 was attributable to the regulations. In addition, two-thirds of the 1971-74 TSPs decline in nonattainment counties occurred from 1971-72, the first year of CAAA enforcement. Consequently, our analysis focuses on this abrupt, one-year improvement in air quality.[9]

Second, below we find evidence that this design may reduce the role of omitted variables bias in analyzing the association between TSPs and infant mortality. Panel B of Figure 2 displays the trends in internal infant mortality rates (IMR) in the same two sets of counties.[10] It shows that before the CAAA was enforced, infant mortality rates were 120-150 deaths per 100,000 births higher in nonattainment counties. Further, attainment and nonattainment counties had similar IMR trends between 1969 and 1971, with attainment counties experiencing a slightly larger decline. However, this pattern is reversed between 1971 and 1972, with the attainment-nonattainment IMR gap narrowing by over 75 deaths per 100,000 births. The figure also reveals that attainment counties had a larger decline in infant mortality between 1972 and 1974.

The correspondence of the trend breaks in TSPs and infant mortality differences between nonattainment and attainment counties in 1972 suggests a causal relationship. Since the patterns after 1972 are less compelling, we probe the case for causality more rigorously in the analysis below. To

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1970.

[9] We probed how industrial polluters may have significantly reduced TSPs emissions in this narrow time frame. Electrostatic precipitators were one of the primary control devices used to abate particulates pollution during this period. The Industrial Gas Cleaning Institute reports that while annual sales of precipitators were approximately $50 million from 1968 through 1970, this figure doubled to $100 million in 1971 and 1972 (White 1984). According to Donald Hug of the Environmental Elements Corporation, a supplier of pollution abatement equipment, an electrostatic precipitator ordered in 1971 could be operating in 1972. We conclude that the large decrease in TSPs emissions between 1971 and 1972 is the plausible result of polluter response to the 1970 CAAA.

[10] We define internal infant mortality to be infant death due to health related causes (i.e., 8th International Classification of Diseases (ICD) codes 001 through 799). This excludes non-health related "external" causes of death such as accidents and homicides (i.e., 8th ICD codes 800 through 999).

maximize the signal-to-noise ratio in TSPs changes and minimize the potential biases from confounding changes in other variables, we focus on the striking one-year changes that occurred between 1971 and 1972. The analysis uses nonattainment status as an instrumental variable for 1971-1972 changes in TSPs. Since federally-mandated regulatory pressure is plausibly orthogonal to changes in infant mortality rates, except through its impact on air pollution, nonattainment status may be a valid instrument. Consistent with this, we find little association between nonattainment status and other observable variables, including parents' characteristics, prenatal care utilization, and transfer payments from social programs.

Further, nonattainment status in 1972 was a discrete function of the annual geometric mean and second highest daily concentrations of TSPs in the regulation selection year, 1970. Thus, the assignment of regulatory status has the feature of a quasi-experimental regression-discontinuity design (Cook and Campbell 1979). If other factors affecting infant mortality are similar for counties just above and just below the regulatory thresholds, then comparing outcome changes in nonattainment and attainment counties with pre-regulation TSPs levels just around the threshold will control for all omitted factors correlated with TSPs. Under this assumption discrete differences in mean outcome changes between nonattainment and attainment counties near the federal ceilings can be attributed to the regulations. The discontinuity in the assignment of regulations provides a valuable opportunity to gauge the credibility of the research design and develop convincing specification tests.

Third, our focus on infant, rather than adult, mortality may circumvent the other important criticisms of previous research. The problem of unknown lifetime exposure to pollution is significantly mitigated, if not solved, by the low migration rates of pregnant women and infants. Thus, we assign TSPs levels to infants based on the exposure of the mother during pregnancy and the exposure of the newborn during the first few months after birth (we focus on deaths within 24 hours, 28 days, and 1 year of birth).

Further, since the mortality rate is higher in the first year of life than in the next 20 years combined (NCHS 1993), it is likely that infant deaths represent a large loss in life expectancy. In contrast, time-series studies of adult and elderly mortality based on daily air pollution fluctuations may be detecting effects among the already very ill and dying. In this case, the actual loss of life expectancy may be much smaller than these studies implicitly suggest.

A final advantage of the design is that it permits a direct analysis of the health benefits of TSPs reductions at levels just above the EPA's maximum allowable concentration. The optimality of the CAAAs depends crucially on the existence and relative magnitudes of the health effects above and near the federal regulatory threshold.

## III. Econometric Methods

Our analysis compares changes in infant mortality rates and TSPs pollution from 1971-1972 for counties that were nonattainment and attainment for TSPs in 1972. Here, we discuss the econometric models used to estimate the infant mortality-TSPs association. For simplicity, it is assumed that the "true" effect of exposure to particulates pollution is homogeneous across infants and over time.

The cross-sectional model predominantly used in the literature on air pollution and health is:

(1) $y_{ct} = X_{ct}'\beta + \theta T_{ct} + \varepsilon_{ct}, \qquad \varepsilon_{ct} = \alpha_c + u_{ct}$

(2) $T_{ct} = X_{ct}'\Pi + \eta_{ct}, \qquad \eta_{ct} = \lambda_c + v_{ct},$

where $y_{ct}$ is the infant mortality rate in county c in year t, $X_{ct}$ is a vector of observed characteristics, $T_{ct}$ is the mean of TSPs across all monitors in the county, and $\varepsilon_{ct}$ and $\eta_{ct}$ are the unobservable determinants of infant mortality rates and TSPs levels, respectively. The coefficient $\theta$ is the "true" effect of TSPs on infant mortality. For consistent estimation, the least squares estimator of $\theta$ requires $E[\varepsilon_{ct}\eta_{ct}]=0$. If there are omitted permanent ($\alpha_c$ and $\lambda_c$) or transitory ($u_{ct}$ and $v_{ct}$) factors that covary with both TSPs and infant mortality, then the cross-sectional estimator will be biased.

With repeated observations over time, a "fixed-effects" model implies that first-differencing the data will absorb the permanent county effects, $\alpha_c$ and $\lambda_c$. This leads to:

(3) $y_{ct} - y_{ct-1} = (X_{ct} - X_{ct-1})'\beta + \theta(T_{ct} - T_{ct-1}) + (u_{ct} - u_{ct-1})$

(4) $T_{ct} - T_{ct-1} = (X_{ct} - X_{ct-1})'\Pi + (v_{ct} - v_{ct-1}).$

For identification, the fixed effects estimator of $\theta$ requires $E[(u_{ct} - u_{ct-1})(v_{ct} - v_{ct-1})]=0$. That is, there are no unobserved shocks to TSPs levels that covary with unobserved shocks to infant mortality rates.

While first-differencing removes permanent sources of bias, it may magnify any attenuation bias due to measurement error. Since our measure of TSPs concentrations is an average across the monitors in

a county, it imperfectly captures individual exposure to TSPs.[11] This mismeasurement will attenuate the estimated TSPs regression coefficient leading to a bias that is proportional to the fraction of the total variation in TSPs that is attributable to measurement error. To the extent that the serial correlation in "true" TSPs exposure is greater than the autocorrelation in the TSPs measurement error, first-differencing the data will increase the magnitude of the attenuation bias.[12]

Now suppose there exists an instrumental variable, $Z_c$, that causes changes in TSPs without having a direct effect on infant mortality rate changes. Such a variable would purge the estimates of the biases due to both omitted variables and measurement error. One plausible instrument is the 1970 CAAA regulatory intervention for TSPs, measured by the 1972 attainment-nonattainment status of a county. Here, equation (4) becomes:

(5) $T_{c72} - T_{c71} = (X_{c72} - X_{c71})'\Pi_{TX} + Z_{c72}\Pi_{TZ} + (v_{c72} - v_{c71})$, and

(6) $Z_{c72} = 1(T_{c70} > \overline{T}) = 1(v_{c70} > \overline{T} - X_{c70}'\Pi - \lambda_c)$,

where $Z_{c72}$ is the regulatory status of county c in 1972, $1(\bullet)$ is an indicator function equal to one if the enclosed statement is true, and $\overline{T}$ is the maximum concentration of TSPs allowed by the federal regulations. Regulatory status in 1972 is a discrete function of 1970 pollution levels.[13]

An attractive feature of this research design is that the reduced-form relations between 1972 TSPs nonattainment status and the two endogenous variables provide direct estimates of the benefits of the regulations. In particular, $\Pi_{TZ}$ from equation (5) measures the 1971-1972 air quality improvement in nonattainment counties relative to the change in attainment counties. In the other reduced-form equation,

(7) $y_{c72} - y_{c71} = (X_{c72} - X_{c71})'\Pi_{yX} + Z_{c72}\Pi_{yZ} + (u_{c72} - u_{c71})$,

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[11] During the 1971-1972 period, the mean and median numbers of monitors in a county are 4.2 and 2, respectively.

[12] However, in our context it is unclear whether the serial correlation in the TSPs measurement error from 1971 to 1972 is smaller or larger than the serial correlation in true TSPs exposure.

[13] For simplicity, we have written (6) as if regulatory status is a function of a single threshold crossing. If $T_{c70}^{avg}$ and $T_{c70}^{max}$ are the annual geometric mean and 2nd highest daily TSPs concentrations, respectively, then the actual regulatory instrument used is $1(T_{c70}^{avg} > 75\ \mu g/m^3$ or $T_{c70}^{max} > 260\ \mu g/m^3)$. Only six counties were nonattainment in 1972 for exceeding the 2nd highest daily concentration threshold, but not the annual geometric mean ceiling. See footnote 7 and the Data Appendix for details on the timing of the CAAA intervention.

$\Pi_{yZ}$ captures the relative improvement in the infant mortality rate in nonattainment counties between 1971 and 1972.

Since the instrumental variables (IV) estimator ($\theta_{IV}$) of the effect of TSPs on infant mortality is exactly identified, it is equal to the ratio of the two reduced-form relations – i.e., $\theta_{IV} = \Pi_{yZ}/\Pi_{TZ}$. Two sufficient conditions for $\theta_{IV}$ to provide a consistent estimate of the TSPs effect are $\Pi_{TZ} \neq 0$ and $E[v_{c70}(u_{c72} - u_{c71})]=0$. The first condition requires that the regulations induced air quality improvements. The second condition requires that transitory shocks to TSPs levels in 1970 are orthogonal to unobserved shocks to infant mortality rates from 1971-1972.[14]

Even if $E[v_{c70}(u_{c72} - u_{c71})] \neq 0$, causal inferences on $\theta$ may be possible by leveraging the regression discontinuity (RD) design implicit in the $1(\bullet)$ function that determines nonattainment status. For example, if $E[v_{c70}(u_{c72} - u_{c71})] = 0$ in the neighborhood of the regulatory ceiling (i.e., 75 $\mu g/m^3$), then a comparison of changes in nonattainment and attainment counties in this neighborhood will control for all omitted variables. Below, we present IV estimates based on all counties and RD estimates in which the sample is limited to counties with TSPs concentrations "near" the 75 $\mu g/m^3$ threshold in the regulation selection year.[15] In the next section, we show that the observable characteristics of nonattainment and attainment counties near the federal TSPs ceiling are very similar.

Another potential source of bias comes from unmeasured state-specific determinants of infant mortality (e.g., generosity of state Medicaid programs, state weather conditions). Since the analysis is at the county level, in some specifications we control for unrestricted state-time effects in infant mortality to absorb all unobservables that vary across states over time. Here, the effect of TSPs is identified using only comparisons between attainment and nonattainment counties within the same state. Figure 3 provides a graphical overview of the location of attainment and nonattainment counties in 1972. A county's shading indicates its regulatory status – light gray for attainment, black for nonattainment, and

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[14] In the simplest case, the IV estimator will be consistent if 1972 nonattainment status is orthogonal to unobserved infant mortality shocks – i.e., $E[Z_{c72}(u_{c72} - u_{c71})]=0$, which is a stronger condition than $E[v_{c70}(u_{c72} - u_{c71})]=0$.

[15] If the relationship between $v_{c70}$ and $(u_{c72} - u_{c71})$ is sufficiently "smooth" at the regulatory ceilings, then causal inference may also be possible by including smooth functions of the selection variable, $T_{c70}$, as controls. Below, we also provide RD estimates that are adjusted for a quadratic in $T_{c70}$.

white for the counties without TSPs pollution monitors. The pervasiveness of the 1972 TSPs regulatory program is evident. In addition to the traditional counties in the Rust Belt and in the South Coast Air Basin around Los Angeles, scores of other counties were designated nonattainment. Importantly, most states contain both attainment and nonattainment counties.

## IV. Data Sources and Summary Statistics

To implement our evaluation strategy, we use county level data on air pollution, infant health, and a number of additional control variables for the 1969-1974 period. Here, we describe these data and provide summary statistics showing that the nonattainment-attainment research design balances the observable characteristics of counties. More details on the data sources and variables are contained in the Data Appendix.

### A. Data Sources

The health outcome variables and a number of the control variables come from the National Mortality Detail Files and National Natality Detail Files, which are derived from censuses of death and birth certificates.[16] We merge the individual birth and infant death records at the county level for each year to create county by year cells. For each cell, the infant mortality rate is calculated as the ratio of the total number of infant deaths of a certain type (age and cause of death) to the total number of births. The mortality rates within 24 hours, 28 days (neonatal), and one year of birth are computed separately for each year. Fatalities that occur soon after birth are thought to reflect poor fetal development. Thus, we use the estimated effects of TSPs on 24-hour and neonatal mortality to probe whether fetal exposure to TSPs may have adverse health effects.

We also calculate separate mortality rates for deaths due to internal health reasons and deaths due to external "non-health" related causes such as accidents and homicides.[17] Internal infant deaths are not

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[16] The Natality Files provide a 100% census of all births in every year from 1969-1974. The Mortality Files provide a 100% census of all deaths in every year except for 1972, in which a 50% random sample of all deaths is provided.
[17] "Internal" and "external" deaths span all possible causes of death. See footnote 10 for the definition of the internal and external categories based on the 8th ICD codes.

disaggregated further for two broad reasons. First, coroners assign about 70% of these deaths to two categories -- "certain causes of perinatal morbidity and mortality" and "congenital anomalies." Second, since the pathophysiological pathways through which TSPs may affect fetal or infant health are largely unknown, there is no a priori basis for dividing these causes of death into ones that are and are not plausibly related to TSPs.[18] However, it seems reasonable to assume that there is no causal pathway linking air pollution to external causes of death. So we use the estimated association between external mortality rates and TSPs pollution as a check on the internal consistency of our findings.[19]

The microdata on the control variables available in the Natality Files are also aggregated into annual county cells. The detailed Natality variables include information on: socioeconomic and demographic characteristics of the parents; medical system utilization, including prenatal care usage (Rosenzweig and Schultz 1983; Institute of Medicine 1985); maternal health endowment, such as mother's age and pregnancy history (Rosenzweig and Schultz 1983; Rosenzweig and Wolpin 1991); and infant birth weight. Since TSPs may affect fetal development, we use infant birth weight as another outcome variable. The Data Appendix describes the Natality variables used in the analysis in detail.

Annual, monitor-level data on TSPs concentrations were obtained by filing a Freedom of Information Act request with the EPA. This yielded annual summary information from the EPA's nationwide network of pollution monitors, including the location of each monitor and their readings. The data also contain information on the annual geometric mean TSPs concentration for each monitor in a county and the number of daily monitor readings exceeding the federal standards in each year. These measures are used to determine the annual nonattainment status of each county (see the Data Appendix for more details). From the data, we also calculated the mean of TSPs concentrations in each county and year, which we use as our measure of TSPs exposure in the analysis.[20]

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[18] See Utell and Samet (1996) for a summary of the evidence on the pathophysiological relationship between TSPs and human health.

[19] That is, the association between TSPs and external infant deaths may result from unobserved secular factors that affect all types of infant death.

[20] Specifically, the annual TSPs concentration used is the weighted average of the annual arithmetic means of each monitor in the county, with the number of observations per monitor used as weights. Some recent research has focused on the effects of PM-10 particles. Most of these studies multiplied TSPs concentrations by a constant factor to obtain PM-10 measures (e.g., Pope, Dockery, and Schwartz, 1995).

In addition to the key infant health and pollution data, we use a variety of other county-level data as controls. Per-capita income data come from the Bureau of Economic Analysis' annual series, which provides the most comprehensive measure of income available at the county level. This file also provides annual, county-level data on per-capita net earnings, the ratio of total employment to the total population, and the ratio of total manufacturing employment to the total population. The Regional Economic Information System file provides annual, county-level data on several different categories of transfer payments. These include separate series for total transfer payments; total medical care payments; public expenditures on medical care for low-income individuals (primarily Medicaid and local assistance programs); income maintenance benefits; family assistance payments, including AFDC; Food Stamps payments; and Unemployment Insurance benefits.

Table 1 presents summary information from 1969-1974 for the 501 counties with available TSPs and infant mortality data in 1970, 1971, and 1972. This is our primary sample in the below analysis. In 1969 and 1973-74, the sample is further restricted to counties with nonmissing data on TSPs and infant mortality in those years.

The top rows of the table show that these counties account for over 60% of the 3.2-3.7 million live births that occur annually in the United States in this period. The next rows present infant fatalities per 100,000 live births, by cause of death. The internal infant mortality rate in 1971 is 1.8 deaths within a year of birth per 100 births, while infant deaths due to external causes are much rarer. In 1971, about 45 and 75 percent of all internal infant deaths within a year of birth occur within the first 24 hours and 28 days of birth, respectively. The table also shows national trends in infant mortality, average TSPs concentrations, and per-capita income across counties. Internal infant mortality rates decreased steadily from 1969-74. TSPs, on the other hand, fell by over 18 percent from 1971-74, but were relatively stable from 1969-71. Per-capita income increased by 8.5 percent from 1971-73, before falling slightly from 1973-74, presumably a result of the recession.

The remaining rows of Table 1 present 1969-74 trends for several of the control variables used in the analysis. Importantly, the magnitude of changes in these variables is small when compared to the TSPs pollution changes that occurred from 1971-72. Our measures of parental background characteristics

suggest that the average socioeconomic status (SES) of parents was declining from 1969-74, as the fractions of births to single, high school dropout, and black mothers increased. On the other hand, the likelihood of a mother receiving prenatal care or having a prenatal visit in the first trimester increased slightly. The percentage of births attributable to teenagers rose, and the share among women aged 35 and over fell.[21] Finally, the share of first-time births increased slightly while the fraction of mothers who had a prior fetal death remained stable.

B. Balancing of Observable Characteristics

Before proceeding, we examine whether the nonattainment instrumental variable is orthogonal to the observable predictors of infant mortality. While it is not a formal test of the exogeneity of the instrument, it seems reasonable to presume that research designs that meet this criterion may suffer from smaller omitted variables bias. First, designs that balance the observable covariates may be more likely to also balance the unobservables (Altonji, Elder, and Taber 2000).[22] Second, if the instrument balances the observables, then consistent inference does not depend on functional form assumptions on the relations between the observable confounders and infant mortality. Estimators that misspecify these functional forms (e.g., linear regression adjustment when the conditional expectations function is nonlinear) will be biased.

Table 2 shows the association of TSPs levels, TSPs changes, and TSPs nonattainment status with other potential correlates of infant mortality using the same sample of counties as in Table 1. Each column of the table presents the differences in the variable means between two sets of counties (with standard errors in parentheses). The first column contains differences in the 1971 levels of the covariates between counties with high (above the median) and low (below the median) TSPs concentrations in 1971. If TSPs levels were randomly assigned across counties, one would expect very few significant differences between the two groups. However, there are significant differences for several key variables, including

[21] Rees, et. al. (1996) find that much of the correlation between poor infant health and mother's age is due to the higher incidence of low birth weight births among teenagers and women 35 and older.
[22] For example, in experimental designs testing for differences in the observable characteristics of the treatment and control groups is used to assess whether the treatment was actually randomly assigned.

mother's race, immigrant status, use of prenatal care, and likelihood of being a teenager. Though not presented in the table, there are also large and significant differences between the county groups in per-capita receipts of income maintenance benefits, family assistance payments, and public expenditures on medical care for low-income individuals (e.g., see Chay, Dobkin, and Greenstone 2003). Overall, these findings suggest that "conventional" cross-sectional estimates may be biased due to omitted variables.

The next two columns of Table 2 perform a similar analysis for 1971-1972 TSPs changes. They contain differences in 1971 variable levels and 1971-72 variable changes between counties that had large (above the median) and small (below the median) reductions in TSPs between 1971 and 1972. These two county groups exhibit significant differences in the 1971 levels of per capita income, mother's immigrant status, prenatal care use, likelihood of being over 34 years old, and pregnancy history. There are also significant differences in the 1971-72 changes in mother's and father's education levels and marginally significant differences in the changes in the fractions of mothers who are teenagers and who had a prior fetal death. Thus, fixed effects models may also lead to biased inference.

The next two columns of the table show the differences in 1971 levels and 1971-72 changes between nonattainment and attainment counties. While nonattainment status is associated with a large and significant reduction in TSPs concentrations, it is uncorrelated with nearly all of the 1971 levels and 1971-72 changes in the covariates, with the only exceptions being per capita income and mother's race in 1971. The differences in the changes in mother's and father's education and in the changes in the incidences of mothers who are teenagers and experienced a previous fetal death are greatly reduced by comparing nonattainment and attainment counties. Also, in contrast to the previous two sets of county group comparisons, nonattainment and attainment counties exhibit little difference in their receipts of transfer payments from social programs (results available from the authors). Thus, it appears that the attainment-nonattainment research design does a better job of balancing the observables.[23]

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[23] It should be noted that the composition of counties who are nonattainment and attainment in 1972 is different from the composition of counties with "high" and "low" mean TSPs in 1971 (i.e., column 1).

In the last two columns, the sample is further restricted to the 176 counties with 1970 geometric mean TSPs concentrations between 60 and 90 $\mu g/m^3$ – i.e., counties with TSPs levels near the TSPs regulatory ceiling in the regulation selection year. It is evident that focusing on nonattainment and attainment counties near the regulatory threshold eliminates all of the significant differences in the covariate levels and changes, including per capita income, mother's race, and the infant mortality rate in the year before CAAA enforcement. While nonattainment status is orthogonal to all of the other covariates, the second row shows that it is still associated with a sharp reduction in TSPs levels between 1971 and 1972 for counties near the regulatory threshold.[24] These results provide reassuring evidence on the quality of the instrumental variables research design used in this study. We probe this further below.

## V. Empirical Results

### A. Cross-Sectional and Fixed Effects Results

First, we replicate the conventional cross-sectional approach to estimating the association between TSPs pollution and infant mortality that is common in the literature. Table 3 presents the regression estimates of the effect of TSPs on the number of internal infant deaths within a year of birth per 100,000 live births for each cross-section from 1969-1974. The final row contains the results for the pooled 1969-1974 data. Column 1 presents the unadjusted TSPs coefficient; column 2 includes the basic control variables available in the Natality data; column 3 includes additional Natality variables and flexible functional forms for the variables; and column 4 further adjusts for per-capita income, earnings, employment, and transfer payments by source.[25] Columns 5 and 6 add unrestricted state effects to the column 2 and column 4 specifications, respectively. The sample sizes and $R^2$'s of the regressions are shown in brackets.

There is wide variability in the estimated effects of TSPs, both across specifications for a given cross-section and across cross-sections for a given specification. While the raw correlations in column 1

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[24] Given the notation in equations (6) and (7), it should be noted that 1972 nonattainment status is orthogonal to the levels of covariates in 1970 as well (results available from the authors).

[25] The control variables included in each specification are listed in the Data Appendix.

are all positive, only those from the 1969 and 1974 cross-sections are statistically significant at conventional levels.[26] Including the basic Natality controls in column 2 reduces the point estimates substantially in most years, even as the precision of the estimates increases due to the greatly improved fit of the regressions. In addition, only two of the estimates in columns 3 and 4 are significant at conventional levels, and one of these has a perverse negative sign. This is also true of the most unrestricted specification in column 6 that also adjusts for state fixed effects.

The largest positive estimates from the cross-sectional analyses imply that a 1-$\mu g/m^3$ reduction in mean TSPs results in roughly 3 fewer internal infant deaths per 100,000 live births, which is an elasticity of 0.14. Overall, however, there is little evidence of a systematic cross-sectional association between particulates pollution and infant survival rates. While the 1974 cross-section produces estimates that are positive, significant and slightly less sensitive to specification, the 1972 cross-section provides estimates that are routinely negative. The sensitivity of the results to the year analyzed and the set of variables used as controls suggests that omitted variables may play an important role in cross-sectional analysis. We suspect that the fragility of the cross-sectional results to changes in specification and sample is likely to apply to the estimation of the relationship between other measures of human health (e.g., adult mortality) and environmental quality (e.g., other air pollutants).

Table 4 presents the fixed effects estimates of the association between mean TSPs and internal infant mortality rates based on 1969-1974 pooled data (first panel) and 1971-1972 first-differenced data (second panel). The columns correspond to similar regression specifications as those in Table 3. The 1971-1972 first differences results provide a baseline for comparison to the subsequent instrumental variables results that use the same samples and control variables. The inclusion of county fixed effects or the use of first-differenced data eliminates the bias in the cross-sectional estimates attributable to time-invariant omitted factors that vary across counties. However, these approaches will be biased if there are unobserved shocks that are correlated with changes in TSPs and infant mortality rates.

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[26] It is worth noting that the comparison of counties with "high" and "low" 1971 TSPs levels in the first column of Table 2 implies an unadjusted TSPs effect of 2.8 (129.8/45.8), which is 75% greater than the estimated correlation for 1971 in Table 3 (1.6). This is consistent with measurement error in TSPs leading to substantive attenuation bias, since the aggregation in Table 2 "averages out" the measurement errors over many counties.

The 1969-1974 panel shows that the inclusion of county-specific intercepts greatly improves the fit of the regressions when compared to the pooled cross-sectional regressions in the last row of Table 3. The raw fixed effects correlation in column 1 is positive and highly significant. However, the estimated TSPs coefficient falls significantly when the analysis controls for the Natality variables, economic conditions, transfer payments, year effects and state-year effects. The 1971-1972 first differences results also suggest little association between changes in TSPs and changes in infant mortality rates.

The fixed effects association between TSPs and infant mortality is small and sensitive to specification, which is consistent with potential biases due to omitted variables and/or measurement error.[27] We conclude that conventional approaches may not provide reliable estimates of the causal effect of TSPs on infant mortality and turn our attention to the instrumental variables design outlined above.

B. The Impact of the 1970 Clean Air Act on Air Quality and Infant Mortality

The instrumental variables estimate of the effect of TSPs is a function of two reduced-form effects: the impact of 1972 nonttainment status on improvements in air quality, and its association with declines in infant mortality. Here, we provide estimates of the air quality and infant health benefits of the 1970 Clean Air Act and present evidence that our design may plausibly identify the causal effects of nonattainment status on both outcomes.

Figure 4 depicts the 1969-1974 time-series of the raw differences in TSPs levels (Panel A) and internal infant mortality rates (Panel B) between counties that were nonattainment and attainment in 1972. Separate series are plotted for three samples: 1) all counties with nonmissing TSPs data, 2) the subset of counties with a 1970 geometric mean TSPs concentration between 50-115 $\mu g/m^3$, and 3) the subset of counties with 1970 TSPs between 60-90 $\mu g/m^3$.[28] The last two samples are restricted to those counties with TSPs concentrations closer to the 75 $\mu g/m^3$ regulation ceiling in the regulation selection year.

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[27] The comparison of counties with "big" and "small" 1971-1972 TSPs reductions in the third column of Table 2 implies an unadjusted TSPs effect of -0.73 (18.8/(-25.6)) for 1971-1972 first differences. This suggests that the small 1971-1972 first-differences estimate in column 1 of Table 4 is not the result of attenuation bias due to measurement error. See footnotes 12 and 26 for more details.

[28] The samples consist of the fixed set of counties with continuous monitor readings from 1969-1974. The numbers of counties are 401, 262, and 141, respectively.

Panel A shows large trend breaks in the nonattainment-attainment difference in TSPs that correspond with the timing of the first year of CAAA enforcement in 1972. There is a clear convergence in TSPs from 1971 to 1972 even for the subset of counties with 1970 TSPs levels close to the regulatory threshold. Among counties with 1970 TSPs between 60-90 $\mu g/m^3$, the 10.5 unit nonattainment-attainment gap in TSPs in 1971 is almost eliminated by 1972 before rebounding somewhat in 1973. For counties with 1970 TSPs between 50-115 $\mu g/m^3$, there is also a significant relative reduction in TSPs from 1971-1972, though the break in trend appears to have started a year earlier than for the other two county groups. On the whole, the timing and location of the TSPs reductions suggest that enforcement of the 1970 CAAA led to improved air quality.

Panel B of the figure shows a striking correspondence between the breaks in trend in infant mortality differences between nonattainment and attainment counties and those in TSPs differences. For the full sample, the sharp break in infant mortality trends from 1971-1972 matches the break in TSPs differences. The differential timing of the relative shift down in infant mortality across the three samples also matches the TSPs patterns. While the full sample and the sample with 1970 TSPs between 60-90 $\mu g/m^3$ show relative mortality reductions from 1971-1972 only, the sample with 1970 TSPs between 50-115 $\mu g/m^3$ exhibits a relative mortality reduction the previous year also. Also consistent with the patterns in Panel A, the sample with 1970 TSPs between 60-90 $\mu g/m^3$ exhibits the largest rebound in relative mortality rates from 1972 to 1973. Below, we show that some of the 1972-1974 rebound in infant mortality differences is explained by differential changes in the observable characteristics of attainment and nonattainment counties after 1972.

Taken together, these plots provide evidence of a direct link between regulation-induced improvements in air quality and reductions in infant mortality rates. First, the timing of the abrupt declines in TSPs and infant mortality correlate remarkably well across all three groups of counties. Further, none of the observable covariates, including per-capita income and transfer payments, exhibits similar trend break patterns in nonattainment-attainment differences. It seems unlikely that any unobserved factors had nonattainment-attainment differences that changed as sharply or abruptly as the differences in TSPs and infant mortality from 1971 to 1972. Based on these raw numbers, a 1-$\mu g/m^3$

reduction in mean TSPs leads to 6-14 fewer internal infant deaths per 100,000 live births from 1971-1972, which implies an elasticity between 0.3 and 0.6. The counties with TSPs concentrations closest to the TSPs regulatory ceiling provide the largest estimate.[29]

Table 5 contains the regression results from estimating reduced-form equations (5) and (7). The first panel shows the association between the 1972 nonattainment indicator and changes in mean TSPs from 1971-1972. The second panel presents the estimated effects of nonattainment status on changes in infant deaths due to internal causes per 100,000 live births. The columns correspond to the same six specifications used in Table 4.[30]

In the first panel, the regulation indicator is associated with a 9-12 $\mu g/m^3$ reduction in TSPs from 1971 to 1972. This represents a 9-12% improvement in air quality in nonattainment counties and is similar in magnitude to the effect sizes shown in Figure 4. The estimates are relatively insensitive to the inclusion of controls for the Natality variables, economic conditions, transfer payments, and unrestricted state-year effects in columns 1-5. The estimate is reduced in the most unrestricted specification in column 6, but is still highly significant. In fact, the F-statistics imply that nonattainment status is the most important (observable) determinant of TSPs changes from 1971 to 1972. The first-stage impact of regulation is powerful and explains almost all of the overall reduction in mean TSPs in the sample. This finding contradicts recent research that contends that the CAAAs failed to improve air quality (Goklany 1999).

The second panel reveals another striking empirical regularity. The 1972 TSPs nonattainment variable is associated with 53-116 fewer infant deaths per 100,000 live births. This implies a 3-6% decline in the infant mortality rate in nonattainment counties and is similar in magnitude to the implied effects in Figure 4. The estimates are also generally significant – four of six are significant at the 5-percent level and a fifth at the 10-percent level – and tend to increase in magnitude as more controls are

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[29] Taken literally, the numbers underlying Figure 4 imply that a unit-reduction in TSPs has a larger marginal effect at TSPs concentrations just above the 75-$\mu g^3$ regulatory ceiling than at higher concentrations.

[30] It should be noted that the specifications for columns 1 and 2 of Table 4 do not control for year effects, whereas columns 1 and 2 of Table 5 do include a constant to absorb year effects in the first differences. For 1971-1972 first-differences, the estimates in columns 1 and 2 of Table 4 that include a constant are –0.40 (0.71) and –0.36 (0.67).

added. This is particularly notable given the unpredictability of changes in infant mortality rates. In column 3, for example, only the coefficients on the indicators for twins or greater birth and mother's race have t-ratios similar in magnitude to the t-ratio for the nonattainment coefficient.[31]

These results imply that the Clean Air Act regulations resulted in substantial reductions in infant mortality in nonattainment counties. The weighted average of the estimates (with weights equal to the inverse of the sampling errors) suggests that relative to attainment counties nonattainment counties had 82 fewer infants deaths per 100,000 births. Multiplying this figure by the 1.52 million births that occurred in nonattainment counties in 1972 implies that 1,300 fewer infants died in 1972 than would have in the absence of the 1970 Act.

Figure 5 presents the raw and regression adjusted trends in differences between attainment and nonattainment counties in TSPs (Panel A) and infant mortality (Panel B) from 1969 to 1974. The graph plots the estimated coefficients on interactions of 1972 nonattainment status with year indictors from regressions that also include year main effects. The Adjust 1, Adjust 2, and Adjust 3 series are based on specifications that respectively use the same controls as in columns 2-4 of Table 5.[32] Panel A of the figure shows that regression adjustment has little effect on the size of the trend break in relative TSPs in nonattainment counties after 1971.

Pancl B shows that regression adjustment also has little effect on the size of the relative infant mortality reduction from 1971 to 1972 and, if anything, tends to increase the measured improvement in nonattainment counties. Covariate adjustment does however have a substantial effect on the levels of the nonattainment-attainment mortality differences in the pre-regulation period, 1969-1971. Importantly, it also reduces the magnitude of the rebound in relative mortality rates from 1972 to 1974, suggesting that some of the rebound is due to a worsening in the observable characteristics of nonattainment counties relative to attainment ones. Appendix Figure 1, which plots the predicted mortality differences between

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[31] Being black and having a twins or greater birth are both associated with higher rates of infant death. The estimated coefficients on almost all of the other variables (e.g., parents' socioeconomic characteristics, mother's prenatal care use and pregnancy history) are statistically insignificant.

[32] The regressions use the pooled 1969-1974 data and restrict the effects of the control variables to be constant over time. The sample is the fixed set of 401 counties with continuous TSPs readings from 1969-1974.

nonattainment and attainment counties based on the Adjust 3 regression specification excluding TSPs, confirms this conclusion.[33] It shows that in the absence of the TSPs changes, the nonattainment-attainment mortality gap would have been predicted to widen over the period due to the general worsening of the relative characteristics of nonattainment counties.

These findings suggest that while the observable controls are strong predictors of infant mortality levels, they cannot account for the sharp mortality change in nonattainment counties from 1971 to 1972. Interestingly, Figure 5 also explains the differences across cross-sections in the estimated TSPs coefficients in Table 3. In 1972, for example, nonattainment counties had higher TSPs levels than attainment counties but lower infant mortality rates once differences in observable predictors are controlled for. This corresponds with the perverse, negative estimates for the 1972 cross-section in Table 3. The nonattainment-attainment differences in TSPs and infant mortality in 1974 also correspond with the systematically positive estimates for the 1974 cross-section. In fact, much of the across-year variation in the cross-sectional estimates in Table 3 appears to be due to the sharp improvement in air quality caused by the Clean Air Act.

Our final assessment of the causal impact of the 1970 Act on air quality and infant mortality exploits the discontinuity in the assignment rule that determines 1972 nonattainment status. Figure 6 graphs the bivariate relations of both the 1971-1972 change in TSPs and the 1971-1972 change in infant mortality with the geometric mean of TSPs levels in 1970 (the regulation selection year). The plots come from the estimation of nonparametric regressions that use a uniform kernel density regression smoother. Thus, they represent a moving average of the raw changes across 1970 TSPs levels.[34] This graphical analysis allows us to test whether there are sharp differences in the outcomes that correspond with the discrete zero-to-one change in the probability that a county was designated nonattainment at the

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[33] Appendix Figure 1 was constructed from two steps: 1) a regression of infant mortality rates on the observables excluding TSPs; and 2) a regression of the predicted infant mortality rates from the first regression on year effects and interactions of the year effects with 1972 nonattainment status. Thus, the figure provides a "single-index" summary of the differences between nonattainment and attainment counties in the observable confounders.

[34] The smoothed scatterplots are insensitive to the choice of bandwidth.

regulatory ceiling (the vertical line in the figure) mandated by the CAAA. Recall, counties with 1970 geometric mean TSPs levels below (above) 75 $\mu g/m^3$ are attainment (nonattainment) in 1972.[35]

For the full set of counties in our primary sample, the figure confirms the regression results in Table 5. Nonattainment counties with 1970 TSPs levels above the regulatory ceiling had much larger reductions in both mean TSPs and infant mortality rates from 1971-1972 than their attainment counterparts with 1970 TSPs below the ceiling. The most striking features of the graph, however, are the clear "trend breaks" in TSPs and infant mortality rate changes at the regulatory threshold. These breaks strongly suggest that the CAAA regulation is a causal factor in improvements in both outcomes for nonattainment counties.

Focusing on the counties with geometric mean TSPs between 50 and 100 $\mu g/m^3$ in 1970, there is a clear association between larger reductions in mean TSPs and greater decreases in infant mortality at the EPA-mandated air quality standard. The correspondence of the trend breaks implies that our research design may identify the casual effect of air pollution on infant mortality through the mechanism of regulation. This "nonparametric" display of the data suggests instrumental variables estimates that are similar in magnitude to the two-stage least squares estimates presented in the next section.

On the other hand, there appear to be secular decreases in infant mortality rate reductions at 1970 TSPs levels well below (30-50 $\mu g/m^3$) and well above (100-150 $\mu g/m^3$) the 75-$\mu g/m^3$ threshold. Thus, IV estimates based on the subsample of counties close to the regulation discontinuity will be larger in magnitude than those based on the entire sample. This could be due to either a nonconstant infant mortality-TSPs gradient or greater differences in the characteristics of nonattainment and attainment counties with 1970 TSPs levels far away from the discontinuity.

Figure 7 provides two checks of the validity of the 1972 nonattainment research design. First, Panel A of the figure empirically examines the validity of the "smoothness" condition discussed above. It plots the infant mortality changes predicted by the observable covariates excluding TSPs changes, $E[(y_{c72}$-

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[35] The six counties that were nonattainment in 1972 for exceeding the daily concentration standard but not the annual geometric mean standard in 1970 are dropped from the analysis. Thus, the sample contains 264 nonattainment counties and 230 attainment counties.

$y_{c71}$)|($X_{c72}$-$X_{c71}$)], along with the actual infant mortality changes from Figure 6, as a function of 1970 TSPs levels. The predicted changes come from a regression that includes the full set of Natality control variables – i.e., column 3 of Table 5.

The figure shows that this index of the non-pollution determinants of infant mortality changes is smooth at the regulatory threshold. By contrast, actual infant mortality rates fall substantially at the threshold, presumably due to the large TSPs reductions. The mortality predictors do appear to explain the decreasing reductions in infant mortality at low 1970 TSPs levels. But for counties with 1970 TSPs greater than 55-60 $\mu g/m^3$, there is little association between predicted mortality changes and pre-regulation TSPs levels. Consistent with the results in Table 2, this finding suggests that attainment and nonattainment counties in the neighborhood of the regulatory ceiling have the same characteristics. In addition, the only observable variable that exhibits a break at the ceiling that corresponds with the infant mortality break is the change in TSPs.

Panel B of Figure 7 plots pre-regulation changes in infant mortality and mean TSPs from 1969-1970 by the geometric mean of TSPs in 1970. This figure provides the following falsification test. Since the 1970 Clean Air Act was not yet in force, there should not be a "trend break" in 1969-1970 changes in the outcome variables near the 75-$\mu g/m^3$ threshold. The graph shows that pre-regulation changes in infant mortality and TSPs have a smooth relation with 1970 TSPs levels at the regulatory ceiling. This is consistent with 1972 nonattainment status being the cause of the 1971-1972 outcome changes. In fact, before the passage of the 1970 CAAA, nonattainment counties had rising TSPs levels relative to attainment counties, which corresponds with the growing gap in infant mortality in the pre-regulation period. These patterns are consistent with the more aggregated patterns in Figure 4.

Taken together, Figures 4-7and Table 5 provide convincing evidence that the 1970 CAAA had significant air quality and infant health benefits. The results imply that the regulation of nonattainment counties in 1972 resulted in a 10 percent reduction in TSPs concentrations and a 4-5 percent decline in infant mortality rates from 1971 to 1972. Further, the nonattainment design allowed us to develop several credible tests of the causality of the CAAA intervention and of the infant mortality-TSPs relation. Tests of this type are not possible in the cross-sectional settings typically used in the literature.

C. Instrumental Variables Estimates of the Effect of TSPs

Table 6 presents the instrumental variables estimates of the effect of TSPs pollution on internal infant mortality rates within one year of birth. Here, the indicator for 1972 nonattainment status is used as an instrument for 1971-1972 changes in TSPs. The first column presents the unadjusted estimate – i.e., the Wald estimate. The remaining columns correspond to specifications that include the same sets of control variables as in Tables 4 and 5.

While the Wald estimate in column 1 implies that a 1-$\mu g/m^3$ decline in TSPs results in 4.4 fewer infant deaths per 100,000 live births, it is not significant at conventional levels. However, when the Natality controls are added in columns 2 and 3, the estimated TSPs coefficient rises to 7.1 and 8.5 and is significant at the 5-percent level in both cases. The TSPs coefficient is not affected when the controls for economic conditions and transfer payments are added in column 4. The specifications in the final two columns include unrestricted state effects, which absorb unobservable differences across states in 1971-1972 changes. Thus, the treatment effect is identified using only comparisons of changes between attainment and nonattainment counties within the same state. In column 5, the TSPs estimate rises to 13.1 and is highly significant. In the most saturated model in column 6, the TSPs coefficient is 15.1 and is less precisely estimated, which is not surprising.[36]

In direct contrast to the cross-sectional and first-differences estimates, the instrumental variables estimates of the TSPs effect are much larger in magnitude and generally significant. The weighted average of the IV estimates (with weights equal to the inverse of the sampling errors) implies that a 1-$\mu g/m^3$ reduction in TSPs is associated with 8.3 fewer infant deaths per 100,000 live births, which is an elasticity of 0.45. This is roughly three times greater than the largest cross-sectional estimate in Table 3 and is similar in magnitude to the effects found in Chay and Greenstone (2003), who estimated a 0.35 elasticity.[37] For the counties in our sample, the internal infant death rate fell by an average of 55 per

[36] The IV estimate (sampling error) from a specification that includes state-time effects and the unrestricted Natality controls in column 3 is 16.8 (7.1), which is significant at the 1-percent level. Thus, among these seven IV estimates, four are significant at the 5-percent level, two are significant at the 10-percent level, and the Wald estimate is significant at the 15-percent level.

[37] Chay and Greenstone (2003) also find that black infant mortality is more sensitive to county-level TSPs reductions than white infant mortality. Although we find similar qualitative results when we stratify our analysis by

100,000 births from 1971 to 1972. Thus, the estimates also imply that the entire reduction in infant mortality from 1971-1972 is due to the 11-unit average decline in TSPs induced by the 1970 CAAA.

The IV estimates are also more stable across specifications than the cross-sectional and first-differences estimates. However, they do tend to increase in magnitude as the observable controls and state-time effects are added to the specifications. This suggests that in the absence of the regulation-induced TSPs reductions, attainment counties had characteristics that favored greater declines in infant mortality relative to nonattainment counties from 1971-1972. This is consistent with the patterns in Figures 5B and 7A. On the other hand, Table 2 and Figure 7A suggest that comparisons between the nonattainment and attainment counties with 1970 TSPs levels in the neighborhood of the regulatory ceiling may reduce both observable and unobservable sources of bias.

Table 7 examines this possibility by presenting instrumental variables estimates for the subsamples of counties with 1970 geometric mean TSPs near the nonattainment threshold. The first two columns show the estimates for the entire sample of counties. The first specification controls for the basic Natality variables while the second further adjusts for unrestricted state-time effects (i.e., columns 2 and 5 in Table 6).[38] The remaining columns present the IV estimates from these specifications for the subsamples of counties with 1970 geometric mean TSPs between 50-115 and 60-90 $\mu g/m^3$.[39]

The first panel of the table contains the results for internal infant mortality rates within one year of birth. The findings are consistent with our hypothesis. As the sample is limited to counties with 1970 TSPs levels closer to the regulatory threshold, the IV estimates tend to increase and are insensitive to the inclusion of state-time effects. The largest estimates come from the 173 counties with 1970 TSPs levels between 60-90 $\mu g/m^3$. They imply that a 1-$\mu g/m^3$ TSPs reduction results in 15-17 fewer infant deaths per 100,000 live births, which is an elasticity of 0.6-0.7.[40] Further, the estimated reduced-form effects of

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infant's race, the sampling errors on the IV estimates for black infants were too large for a reliable conclusion.

[38] These specifications were chosen for two related reasons. First, while the estimates are insensitive to including additional controls to these specifications, their sampling errors increase. Second, the decrease in precision is exacerbated when the analysis is limited to the smaller numbers of counties with 1970 TSPs levels just above and below the regulatory threshold.

[39] In each case, nonattainment counties account for about half of the sample. Picking different subsamples (e.g., 45-105 $\mu g/m^3$, 50-100 $\mu g/m^3$, etc.) leads to nearly identical results.

[40] The unadjusted Wald IV estimate (sampling error) for this subsample is 12.8 (8.5). The corresponding IV

nonattainment status are significant at the one-percent level for the change in TSPs and at the five-percent level for the change in infant mortality rates (results available from the authors). We conclude that TSPs air pollution appears to have a causal impact on infant survival rates in the first year of life.

D. Evidence on the Pathophysiologic Mechanism

We now attempt to shed light on the pathophysiologic mechanism that underlies the TSPs-infant mortality relation.[41] In particular, we examine whether fetal exposure during pregnancy may account for a portion of the harmful effects of TSPs air pollution. It is thought that most fatalities that occur during the perinatal period – variously defined as beginning with completion of the 20th to 28th week of gestation and ending 7 to 28 days after birth – can be attributed to poor fetal development. Thus, the estimated effect of TSPs reductions on death rates within 28 days and 24 hours of birth may provide evidence on this potential biological pathway. Moreover, there is strong evidence that maternal cigarette smoking can retard fetal development and reduce the birth weight of infants (Sexton and Hebel 1984, Torelli 2000). Consequently, we also examine the effects of TSPs changes on infant birth weight.[42]

The second and third panels of Table 7 report the estimated effects of TSPs on the 28-day (neonatal) and 1-day mortality rates, respectively, based on the same specifications as in the first panel. The numbers in brackets are equal to the ratio of the panel's estimated TSPs coefficient to the 1-year mortality coefficient multiplied by 100. These represent the fraction of the overall infant mortality effect of TSPs that can be attributed to deaths soon after birth.

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estimates (sampling errors) for the 120 counties with 1970 geometric mean TSPs between 65-85 $\mu g/m^3$ are 17.6 (15.2), 20.3 (17.1), and 28.6 (28.9). Although nonattainment counties had a 8-unit greater decline in TSPs than attainment counties in the 65-85 $\mu g/m^3$ subsample, the first-stage coefficient on the 1972 nonattainment variable is not significant at conventional levels.

[41] There exists little evidence in the biological literature on the causal pathways through which air pollution impacts human health (Utell and Samet 1996) and almost no evidence on how it might affect infant health. Controlled experiments on animals ranging from guinea pigs to monkeys provide the strongest evidence on a potential mechanism. These studies find that air pollution exposure can cause a constriction of the bronchial system that impairs lung functioning (Amdur 1996). Although consistent with some non-experimental evidence for humans, it is not clear that these findings can be extrapolated to human adults or infants.

[42] With daily TSPs data and linked individual-level natality and mortality data, in principle one might examine whether abrupt changes in TSPs concentrations in particular months of a pregnancy have differential impacts on birth weight and mortality. Unfortunately, such data are not available for the years covered in this study. Machine-readable data on fetal deaths are also unavailable for this period.

For neonatal mortality, the estimates in the first two columns, based on all counties, imply that a 1-$\mu g/m^3$ decline in TSPs results in 4.2-6.6 fewer deaths within one month of birth (per 100,000 births). Though the estimates are only significant at the 15-percent level, they suggest that 50-60 percent of the effect of TSPs on infant mortality is due to reductions in neonatal mortality. As the analysis focuses on the subsets of counties with 1970 TSPs levels near the regulatory ceiling, however, the estimated effects on neonatal mortality increase monotonically. Among counties with 1970 TSPs levels between 60-90 $\mu g/m^3$, the estimated neonatal mortality effect rises to 12.5, and is significant at the 8-percent level though based on many fewer counties. Further, the estimate is insensitive to adjustment for unrestricted state-time effects, and its standard error increases by relatively little given the small sample size.

The estimates in the final two columns imply that 73-82 percent of the effect of regulation-induced declines in TSPs near the nonattainment discontinuity can be attributed to reductions in neonatal deaths. This result corresponds with the finding of Chay and Greenstone (2003) that roughly 80 percent of the overall effect of recession-induced TSPs changes is due to neonatal mortality reductions. It is also consistent with the possibility that maternal exposure to TSPs during the gestation period may affect fetal development and health.

On the other hand, the third panel of the table shows that 24-hour mortality rates have little statistical association with regulation-induced changes in TSPs. The point estimates are smaller in magnitude than the sampling errors and imply that reductions in one-day mortality account for, at most, 22 percent of the overall improvement in infant mortality. This result differs somewhat from Chay and Greenstone (2003), who find significant one-day mortality effects that account for roughly 60 percent of the overall decline in infant mortality. While not significant, the point estimates for the counties bordering the regulatory discontinuity in the final two columns are similar to those in Chay and Greenstone (2003). On net, however, these results leave open the possibility that infant exposure to particulates pollution in the days immediately after birth may partially drive our estimated effects.

A criticism of research on air pollution and adult mortality is that temporarily elevated pollution levels may just hasten the death of adults who are already dying, implying minimal loss in life expectancy. If this phenomenon, also known as "harvesting", is important in our context, then the

association between TSPs reductions and infant death should disappear as one examines longer periods after birth. Table 7 shows, however, that the estimated effects of TSPs increase as the period since birth lengthens. This is inconsistent with harvesting driving our findings and suggests that the regulation-induced decline in TSPs had long-run effects on life expectancy.

To further address the possibility that the pathophysiologic mechanism is related to fetal exposure, we estimated the association between the regulation-induced TSPs changes and infant birth weight using the same specifications as in Tables 6 and 7 (results available from the authors). We found little systematic association between TSPs and the incidences of either low (less than 2500 grams) or very low (less than 1500 grams) birth weights. The estimates were small in magnitude and insignificant at conventional levels.

These findings, which are very similar to those documented in Chay and Greenstone (2003), imply that TSPs pollution has a much larger impact on infant mortality than on birth weight.[43] To the extent that birth weight is a perfect index of health at birth, this would suggest that postnatal exposure, and not fetal exposure, to TSPs has the most harmful effects on infants. However, Almond, Chay, and Lee (2002) provide evidence that birth weight may not be as reliable a proxy for health at birth as previously thought.[44] Since "conditions arising in the perinatal period" are the leading cause of infant death during our time frame, we conclude that fetal exposure may still be a plausible biological pathway. Further, these results contrast sharply with the documented effects of maternal cigarette smoking on infant health and suggest that TSPs pollution works through a different causal mechanism.[45]

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[43] The estimated effect of TSPs on infant mortality falls very little when controls for average birth weight and eight birth weight category dummies are included in the specifications in Table 7. This implies that most of the effect of TSPs on infant mortality is independent of their effect on birth weight.

[44] Almond, Chay, and Lee (2002) provide evidence that the 5-minute APGAR – a diagnostic test administered immediately after birth that scores newborn's heart rate, respiratory effort, muscle tone, reflex irritability, and color – may be a more reliable proxy for health at birth than birth weight. Unfortunately, the Natality files do not contain data on APGAR scores until the 1978 survey year. Chay and Greenstone (2003) find that TSPs reductions during the 1980-1982 recession have much larger impacts on APGAR scores than on birth weight.

[45] Torelli (2000) finds that while maternal smoking has a strong association with reduced birth weight, it is uncorrelated with infant mortality after adjustment for confounding factors. Cigarette smoking during pregnancy is thought to slow fetal growth by depriving the fetus of oxygen.

Before proceeding, we note that our estimates of the birth weight effects of TSPs may be biased down due to censoring. Specifically, if declines in TSPs also reduce the likelihood of a miscarriage or stillbirth, then conditioning on fetuses that survive to birth will lead to selection bias. The birth weight estimates will be understated if a disproportionate number of the marginal fetuses that survive are in the low end of the birth weight distribution. Similarly, our estimates of the mortality effects may also be understated, since the sample of live births could include a higher fraction of infants with low survival probabilities.

E. Tests of the Internal Validity of the Findings

This study has used comparisons between attainment and nonattainment counties to estimate the causal effect of TSPs on infant mortality. However, as is always the case with a non-experimental design, there is a form of unobserved heterogeneity that can explain the findings without a causal interpretation. In addition to the efforts presented above, we probed the robustness of the estimates in a number of other ways but found little evidence that undermines our basic conclusions. Table 8 presents the results of some of these validity checks.

First, there is no obvious causal pathway linking air pollution to external causes of infant death, such as accidents and homicides. Thus, we use the estimated effects of TSPs changes on external infant mortality rates to evaluate the internal consistency of the findings above. While a weak association does not prove that the estimated effects of TSPs on infant mortality are causal, a significant, positive relation would suggest that the analysis is biased by omitted factors that coincided with the regulation-induced TSPs reductions from 1971-72.

The first panel of Table 8 presents instrumental variables estimates of the impact of TSPs changes on external infant mortality rates. The specifications and sample restrictions are identical to those in Table 7. All six columns of the table show a small and insignificant negative correlation between regulations-induced TSPs changes and external mortality rates. These results provide no evidence that our estimates in Tables 6 and 7 are the result of spurious relations.

Second, an alternative explanation for our findings is that the regulation-induced changes in TSPs coincided with unobserved improvements in medical technologies specific to newborns.[46] This explanation would only be valid if, for example, the supply of medical care facilities for neonates in counties above the regulatory threshold was expanded or upgraded between 1971 and 1972 relative to the supply in counties below the threshold. In an effort to control for this possibility, we collected data on the number of hospitals with "premature nurseries" in 1971 and 1972 for each of the counties in our sample. These data were hand-entered from the 1972 and 1973 editions of the American Hospital Association Guides to the Health Care Field.[47] To the best of our knowledge, these measures of neonatal medical technology have not been previously collected for this period.

The second panel of Table 8 contains the estimated effects of TSPs on infant mortality from instrumental variables specifications that add the 1971-1972 change in the county-level number of "premature nurseries" to the controls used in Table 7. The coefficient estimates and their standard errors are nearly identical to those in Table 7. This finding is robust to also controlling for the 1971 and 1972 levels of "premature nurseries" separately. If the number of premature nurseries is a valid proxy for neonatal medical technologies, then it is evident that this paper's results are not due to changes in the availability of these technologies.

Third, our implementation of the regression discontinuity design in Table 7 relies on the assumption that the omitted variables are "held constant" in the neighborhood of the regulatory threshold. Another possibility is that the unobservables change "smoothly" in this neighborhood. In this case, it may be possible to control for these unobservables by including smooth functions of the geometric mean of TSPs in the regulation selection year as covariates. Here, the instrumental variables estimate of the effect of TSPs is identified only by the discrete changes that occur at the nonattainment threshold.

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[46] See Cutler and Meara (1999) on the importance of improvements in medical technology in reducing infant mortality rates among low weight births.

[47] The 1972 (1973) guide reports information on the characteristics of hospitals during the 12-month period ending September 30, 1971 (1972). The guides define a premature nursery as, "A separate facility used exclusively for the care of infants whose birth weight is 2500 grams or less."

The third panel of Table 8 presents IV estimates from specifications that add the geometric mean of TSPs in 1970 and its square to the controls used in Table 7. The estimates of the TSPs effects are generally larger than the comparable estimates in Table 7. However, due to the large increase in the sampling errors, none of the estimates would be judged to be statistically significant at conventional levels. While the imprecision of the estimates precludes strong conclusions, it underscores that this specification is very demanding of the data and may be too flexible. Nevertheless, these results are consistent with our previous findings of a robust effect of regulation-induced TSPs changes.[48]

Fourth, we obtained a second measure of TSPs-specific regulatory pressure. In August 1973, the EPA published a report containing the particulates "Priority Classifications" for Air Quality Control Regions (AQCRs) in the United States (U.S. EPA 1973). The country was divided into 247 AQCRs, implying an average of over 12 counties in each AQCR. A Priority Classification of I or IA for particulate matter indicates that TSPs concentrations are "most severe." It is likely that the I and IA areas were subject to greater regulatory scrutiny than the remainder of the country. Consequently, we created an alternative regulation variable that is equal to 1 if the county is located in a TSPs Priority I or IA AQCR and 0 otherwise.[49] It should be noted that this regulatory measure is less than ideal for our purposes since it was published in 1973, the year after our 1971-1972 evaluation period. Nonetheless, when this variable is used as an instrument, we find estimated TSPs effects that are greater than those in Table 6 (results available from the authors). However, since there are nearly 3-times fewer AQCRs than counties, the sampling errors increase substantially.

We also performed several other robustness checks. These include: controlling for lagged values of the infant mortality rate; controlling for the 1971 levels of the variables instead of the 1971-72 changes in order to avoid adjusting for "post-treatment" factors; using TSPs measures that are based only on the monitoring sites (defined by longitude and latitude) that were in operation in <u>both</u> 1971 and 1972; and

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[48] The estimates from specifications that include a cubic in the geometric mean of 1970 TSPs are qualitatively similar, but generally have much larger sampling errors.

[49] See the Data Appendix for more details on AQCRs and the determination of our measures of regulation.

adjusting for a full set of AQCR fixed effects. Our qualitative findings are robust to all of these specification checks.

Finally, it is still possible that the TSPs nonattainment regulations caused changes in unmeasured determinants of infant mortality rates, such as other air quality indices.[50] In this case, the instrumental variables estimates will represent the combined effect of changes in TSPs and other air pollutants. Even in this situation, however, our results may still provide the "true" reduced-form effect of nonattainment status on infant mortality. In particular, any competing explanation must exhibit similar "trend breaks" to those shown in Figures 5B and 6 in order to be viable. This would seem to rule out many alternative hypotheses for the infant mortality reductions associated with 1972 nonattainment status.

## VI. Estimates of the Economic Benefits of the Clean Air Act Regulations

Overall, it appears that the 1970 Clean Air Act Amendments resulted in substantial health benefits for infants during the first year that it was in force. Here, we monetize these health improvements and discuss their implications for the optimal design of particulates regulations.

As discussed above, the results imply that roughly 1,300 fewer infants died in 1972 than would have in the absence of the 1970 Act. When a statistical life is valued at $1.6 million to $8.5 million ($1997), this reduction in infant mortality is worth approximately $2.1-11.1 billion ($1997).[51] To the extent that this reduction in TSPs was permanent (recall Figure 1), these benefits would have accrued in each of the 30 years since then. In this case and assuming a 5% discount rate, the present discounted value of these health improvements ranged between $34-180 billion ($1997). Since this calculation ignores the other health and aesthetic benefits associated with lower pollution levels, it is likely to

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[50] In the process of abating TSPs, industrial polluters may also reduce their emissions of other forms of air pollution that affect infant mortality. Data limitations preclude us from exploring this possibility rigorously. In 1971 and 1972 only 10 counties were monitored for all of the other pollutants regulated under the 1970 CAAAs. Further, the numbers of counties monitored for TSPs and just one of the other regulated pollutants are 75 for carbon monoxide, 14 for ozone, 64 for sulfur dioxide, and 47 for nitrogen dioxide.

[51] Viscusi's (1993) review of the literature suggests that the value of a statistical life ranges from $3.5 - $8.5 million ($1997), but recent research by Ashenfelter and Greenstone (2001) indicates that it may be less than $1.6 million ($1997). Notably, these estimates are based on the risk of death among adults. There is an active line of research on how the value of a statistical life varies over the life-cycle.

understate the total economic value of the regulation-induced TSPs reductions.[52] Further, it ignores the benefits of any further reductions in TSPs caused by the Clean Air Act limits in later years. Of course, a full cost-benefit analysis also requires precise information about the costs of these regulations.[53]

This study's results may also be informative about the form of the optimal particulates standard. As discussed in the introduction, the EPA's standard of 75 $\mu g/m^3$ for annual geometric mean TSPs concentrations was chosen to "protect the public health". The optimality of this standard depends on how the infant mortality-TSPs gradient and the marginal cost of abating TSPs vary with the ambient concentration of TSPs. Here we discuss the former and leave the latter for future research.

Taken literally, Figure 6 implies that although there are significant health benefits to a reduction in TSPs at very high concentrations, the marginal benefit of a unit change is greater at TSPs levels below 100 $\mu g/m^3$. Specifically, Table 7 shows that a unit reduction in TSPs results in about 15 fewer infant deaths per 100,000 live births at TSPs concentrations slightly above 75 $\mu g/m^3$. Although this paper's research design does not allow for identification of the infant mortality-TSPs gradient below this threshold, Chay and Greenstone (2003) document that TSPs have substantial effects on infant health at concentrations below 75 $\mu g/m^3$. This suggests that a federal air quality standard below 75 $\mu g/m^3$ could have provided greater health benefits to the public.

## VII. Conclusion

We use the air quality improvements induced by the Clean Air Act Amendments of 1970 to estimate the effects of particulates pollution on infant mortality. The federal air pollution regulations are associated with sharp reductions in both TSPs pollution and infant mortality rates in the first year that the 1970 CAAAs were in force. We estimate that a one percent decline in TSPs results in a 0.5 percent decline in the infant mortality rate. Most of the reduction in infant mortality is driven by a decline in deaths occurring within one month of birth, suggesting that fetal exposure during pregnancy is a

[52] Chay and Greenstone (2000) use a hedonic analysis to value a similar reduction in mean TSPs associated with nonattainment status in the mid-1970s. They calculated a $65 billion ($1997) willingness-to-pay for this change.
[53] See Henderson (1996), Becker and Henderson (2000), and Greenstone (2002) on the costs of the Clean Air Act Amendments in the manufacturing sector.

biological pathway. The results are robust to a battery of tests of the validity of the estimates. Most importantly, there are discrete differences in TSPs and infant mortality changes between counties just above and just below the legislated air quality standard, suggesting that the regulations are a causal factor.

This study provides direct and easily interpreted estimates of the air quality and infant health benefits of the 1970 Act. The results imply that TSPs have substantial effects on infant health at concentrations near the EPA-mandated air quality standard and that roughly 1,300 fewer infants died in 1972 than would have in the absence of the Act. Further, this study's findings cross-validate the findings of our earlier work examining the recession-induced pollution reductions of the early 1980s (Chay and Greenstone 2003).

It has been hypothesized that fine particles have more pernicious effects on human health than large particles. Due to its biological plausibility, in recent years the EPA has shifted its focus away from the regulation of TSPs (defined as all particles with diameters less than 100 μm) and toward the regulation of finer particles. Specifically, the particulate matter standard was changed to apply to particles less than 10 μm in diameter ($PM_{10}$) in 1987 and then to particles less than 2.5 μm in diameter ($PM_{2.5}$) in 1997. Since the EPA did not begin to monitor fine particles until the mid-1980s, this study cannot determine the relative contributions of small and large particles to infant mortality rates.

However, the CAAA regulations were focused on industrial and fuel-combustion related emissions, which are disproportionately comprised of small particles (see U.S. EPA, 1983). Thus, the design in this paper may be utilizing changes in fine particulates pollution, though this is speculative. Future work should focus on resolving this question. Any evidence on this may also be helpful in developing a credible theory of the biological pathways through which air pollution impacts infant health (e.g., how airborne particluates may affect fetal development before birth).

## DATA APPENDIX

### Variables from 1969-1974 National Mortality Detail Files

The 1969-1974 National Mortality Detail Files are an annual census of deaths in the U.S, derived from the Standard Certificate of Death. The data contain the universe of deaths and information on the deceased's county of residence, race, gender, age at death, and cause of death. Since infant deaths cannot be directly linked to individual births for this period, the microdata are aggregated into race-gender-age at death by county of residence cells. The total number of infant deaths (deaths within 1-day, 28 days, and 1-year of birth) within a demographic-county cell is used as the numerator for the infant mortality rate.

### Variables from 1969-1974 National Natality Detail Files

The 1969-1974 National Natality Detail Files are an annual census of births in the U.S, derived from the Standard Certificate of Live Birth. Each file contains the universe of births and information on the county of residence of the mother, socioeconomic and demographic characteristics of the parents, the mother's usage of medical services, the health endowment and medical history of the mother, and the infant health endowment. The microdata on births are aggregated into race-gender by maternal county of residence cells. The total number of births within a demographic-county cell is used as the denominator for the infant mortality rate.

The following variables are included as controls in specifications labeled "Unrestricted Natality" in the tables. The word "indicator" refers to an indicator variable at the individual-level. The cell observation contains the fraction of individual observations for which the indicator is one.

Socioeconomic and Demographic Characteristics

*Mother*

county of residence
continuous years of education
indicators for years of education <12, =12, 13-15, 16+
marital status indicator

*Father*

continuous years of education
indicators for years of education <12, =12, 13-15, 16+
age

*Infant*

racial indicators for white, black, and other birth
indicator for gender of birth
*not* a singleton indicator (twins or greater birth)

Medical System Utilization

month of first prenatal care visit
first prenatal care visit in months 1 or 2 of pregnancy, indicator
first prenatal care visit in month 3, indicator
first prenatal care visit in months 4, 5, or 6, indicator
first prenatal care visit in months 7, 8, or 9, indicator
no prenatal care, indicator
delivery outside of hospital, indicator
physician present at delivery, indicator

Maternal Health Endowment and Medical History

age of mother

indicators for mother's age 10-14, 15-19, 20-24, 25-29, 30-34, 35-39, 40+
1 previous live birth, indicator
2 or more previous live births, indicator
1 previous fetal death, indicator
2 or more previous fetal deaths, indicator
last pregnancy resulted in live birth, indicator
last pregnancy resulted in fetal death, indicator
indicators for 1-11, 12-17, 18 or more months since termination of last pregnancy
indicators for 1-11, 12-17, 18 or more months since last live birth
indicators for 1-11, 12-17, 18 or more months since last fetal death

The following variables are included in specifications labeled "Basic Natality Variables" in the tables: indicators for race and gender of infant, mother's education, father's education, age of mother, marital status of mother, indicators for month of first prenatal care visit, indicator for no prenatal care

We also use the Natality data to examine the following birth weight outcomes for infants:
continuous birth weight
indicators for birth weight < 1000 grams, 1000-1499, 1500-1999, 2000-2499, 2500-2999, 3000-3499, 3500-3999, 4000-4499, 4500+

**Total Suspended Particulates (TSPs) Pollution Data**

The data on suspended particulates pollution levels in each county were obtained by filing a Freedom of Information Act request with the EPA. This yielded the *Quick Look Report* data file, which is derived from the EPA's *Air Quality Subsystem* (AQS) database. This file contains the universe of recordings of TSPs pollution concentrations from each of the EPA air monitors that were located throughout the U.S. during the period of interest, as well as the location of each monitor. For each county, the annual concentration of TSPs used in this study is the weighted average of the annual arithmetic means of each monitor in the county, with the number of observations per monitor used as weights. A monitor observation is the concentration recorded over a 24 hour period.

Our analysis relies on the presumption that the TSPs concentration readings used accurately reflect the "true" exposure of individuals to TSPs. To preclude the possibility that counties or states place monitors to fabricate the appearance of favorable pollution concentrations, the Code of Federal Regulations contains very precise criteria that govern the siting of a monitor.[54] Among the most important criteria is that the monitors capture representative pollution concentrations in high population areas. Moreover, the EPA must approve the location of all monitors and requires documentation that the monitors are actually placed in the approved locations.

**Nonattainment Data**

We attempted to obtain a list of the counties where the EPA applied the TSPs nonattainment designation (and the accompanying regulations) in the first year(s) that the 1970 CAAAs were in force. Despite extensive examinations of publicly available documents and conversations with EPA personnel, we were unable to locate such a list. Consequently, we developed a rule for assigning counties 1972 TSPs attainment-nonattainment designations based on our understanding of the EPA's selection rule in that year. This subsection describes why our primary measure of regulation is defined at the county-level and then explains our assignment rule for 1972.

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[54] This discussion results from the Code of Federal Regulations (CFR) 1995, title 40, part 58 and a conversation with Manny Aquilania and Bob Palorino of the EPA's District 9 Regional Office.

A County-Based Definition of Nonattainment Status

The determination of the geographic area at which the nonattainment regulations were applied is the first step in the development of our nonattainment database. The CAAAs required the division of the country into Air Quality Control Regions (AQCR). This led to the creation of 247 AQCRs, meaning that on average there are roughly 12 counties in each AQCR. Statutorily, each state is required to submit to the EPA a plan for the implementation and enforcement of the NAAQS within each AQCR or "portions thereof" (Reitze Jr. 2001, p. 50). This language is ambiguous as to whether nonattainment designations were applied at the AQCR level or a smaller geographic unit, such as the county.

We chose to assign attainment-nonattainment status at the county level. This assumes that the states designed their SIPs to reduce TSPs concentrations in the counties of an AQCR that exceeded the NAAQS and considered the remaining counties in an AQCR as attainment areas. We believe that this is a reasonable assumption, because it requires that states and the EPA attempted to achieve the NAAQS in the most cost efficient manner—that is, they only applied the more costly nonattainment regulations in the counties within an AQCR that exceeded the NAAQS. Further, the EPA began to annually publish the attainment-nonattainment status of each U.S. county in 1978, making it apparent that nonattainment status officially varied at the county-level in that year and all subsequent ones. We believe that it is reasonable to presume that the nonattainment regulations were also applied at the county-level in earlier years.

Interestingly, there is substantial variation in our 1972 county-level TSPs nonattainment designations within AQCRs. The intra-AQCR correlation in 1972 TSPs nonattainment status is only 0.21. Despite this intra-AQCR variation, we find that our basic results are insensitive to the use of AQCR-based definitions of nonattainment status. The "Tests of Internal Validity" subsection discusses these results further.

The Assignment Rule for 1972 County-Level TSPs Attainment-Nonattainment Status

The dates associated with the passage and enforcement of the 1970 CAAAs guided our determination of the 1972 TSPs attainment-nonattainment designations. Richard Nixon signed the 1970 CAAAs on December 31, 1970. Four months later on April 30, 1971, the EPA announced the final publication of the NAAQS that specified the national TSPs standards. On August 14, 1971, the EPA published, "Requirements for Preparation, Adoption, and Submittal of Implementation Plans," in the Code of Federal Regulations (CFR). This set forth how states were to write their SIPs to achieve compliance with the NAAQS by 1975. Finally, the SIPs were due to the EPA in January, 1972. Appendix Table 1 summarizes these dates.

These dates have two implications for our analysis. First, it is evident that the nonattainment regulations could not be enforced until 1972 as they were not written until January, 1972. This is supported by an EPA publication that states that any improvements in air quality through 1971 are "most probably" not the result of the CAAAs (U.S. EPA 1973, p. 2-2). Second, we suspect that the states ascertained the identity of the counties with TSPs concentrations above the NAAQS while writing their SIPs during the second half of 1971. We assume that they must have relied on the available TSPs concentrations data, which was from 1970. Consequently, we treat 1972 as the first year that the CAAAs were in force and assume that 1970 TSPs concentrations were used to determine TSPs attainment-nonattainment designations for 1972.

To implement this selection rule, we use data from the same network of TSPs monitors used by the states and the EPA in this period. We designate a county as 1972 TSPs nonattainment if the monitor with the highest recorded 1970 concentration in that county exceeds either of the TSPs thresholds specified in the NAAQS. All other monitored counties are attainment. We use the readings from the monitor with the highest concentration, rather than an average across monitors, to provide a "liberal" definition of nonattainment status. This is because we assume that the states and the EPA felt pressure to meet the 1975 deadline for compliance with the NAAQS and wanted to have a margin for error. It is noteworthy that the estimated effects of TSPs on infant mortality rates are generally smaller but still statistically significant when the average across all monitors within a county is used to determine nonattainment status.

**County-Level Per-Capita Income Data**

Ideally, data would be available on the incomes of women giving birth in each county. In its absence, the analysis uses the Bureau of Economic Analysis' annual county-level series on per-capita income (deflated to $1982-84). It is "the sum of wage and salary disbursements, other labor income, proprietors' income with inventory valuation and capital consumption adjustments, rental income of persons with capital consumption adjustment, personal dividend income, and transfer payments to persons, less personal contribution for social insurance" (Bureau of Economic Analysis 1994). This is the most comprehensive measure of income available at the county level and is superior to less broad measures, such as labor income, which do not capture all of the resources available to individuals.

We also have annual, county-level data on per-capita net earnings, the ratio of total employment to the total population, and the ratio of total manufacturing employment to the total population.

**County-Level Data on Transfer Payments**

From the Regional Economic Information System (REIS), we obtained county-level information on medical transfer payments and payments for income maintenance and unemployment insurance. The analysis controls for each of the following payment sub-categories, in terms of per-capita payments (in $1982-84):

Total transfer payments per-capita
Total medical care payments per-capita
Public assistance medical care payments for low income individuals (primarily Medicaid and state and local government general assistance medical programs)
Income maintenance benefit payments per-capita
Family assistance payments per-capita (includes AFDC)
Food stamps payments per-capita
Unemployment Insurance (UI) benefit payments per-capita

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Table 1: Sample Statistics, 1969-1974

| | 1969 | 1970 | 1971 | 1972 | 1973 | 1974 |
|---|---|---|---|---|---|---|
| Number of Counties in Sample | 412 | 501 | 501 | 501 | 495 | 489 |
| Total Births in Sample | 2,114,018 | 2,340,590 | 2,196,180 | 1,994,530 | 1,902,045 | 1,903,117 |
| Total Births in U.S. | 3,600,206 | 3,737,800 | 3,563,548 | 3,266,235 | 3,146,125 | 3,170,631 |
| Fatalites Per 100,000 Live Births | | | | | | |
| Internal Causes | | | | | | |
| At 1 Day | 927.0 | 881.3 | 810.2 | 771.8 | 708.9 | 654.1 |
| At 1 Month | 1,549.6 | 1,483.2 | 1,386.2 | 1,335.9 | 1,268.6 | 1,201.2 |
| At 1 Year | 1,986.8 | 1,899.1 | 1,808.0 | 1,752.3 | 1,678.1 | 1,597.3 |
| External Causes | | | | | | |
| At 1 Year | 65.9 | 61.1 | 64.5 | 57.7 | 58.4 | 49.8 |
| At 1 Year All Causes, by Race | | | | | | |
| Blacks | 3,232.6 | 3,032.0 | 2,828.7 | 2,786.2 | 2,590.5 | 2,551.7 |
| Whites | 1,753.2 | 1,680.6 | 1,602.9 | 1,533.9 | 1,489.6 | 1,400.3 |
| Mean County-Level Pollution, Income, and Employment-Population Ratio | | | | | | |
| TSPs Concentration | 93.1 | 94.2 | 92.4 | 81.3 | 78.3 | 75.6 |
| Per Capita Income ($1982-84) | $11,320 | $11,329 | $11,442 | $11,991 | $12,423 | $12,262 |
| Total Employment/Population | 49.8 | 48.1 | 47.3 | 48.2 | 50.0 | 50.4 |
| Mean Parental Demographic and Socioeconomic Characteristics | | | | | | |
| % Mother H.S. Dropout | 17.2 | 18.5 | 18.8 | 18.9 | 19.5 | 19.0 |
| Mother's Years of Education | 12.1 | 12.1 | 12.1 | 12.1 | 12.1 | 12.2 |
| Father's Years of Education | 12.6 | 12.6 | 12.6 | 12.7 | 12.8 | 12.8 |
| % Single Mother | 7.3 | 7.5 | 8.2 | 9.1 | 9.7 | 9.7 |
| % Black | 16.3 | 16.6 | 17.5 | 18.0 | 18.1 | 17.9 |
| % Foreign-born | ----- | 8.9 | 9.4 | ----- | 10.6 | 11.2 |
| Mean Medical Services Utilization | | | | | | |
| % No Prenatal Care | 1.59 | 1.49 | 1.40 | 1.36 | 1.35 | 1.30 |
| % Prenatal Care in 1st Trimester | 55.1 | 55.3 | 56.0 | 57.2 | 60.2 | 61.3 |
| Number of Prenatal Visits | ----- | ----- | ----- | 9.5 | 9.8 | 10.0 |
| Mean Maternal Health Endowment | | | | | | |
| % Teenage Mother | 16.6 | 16.9 | 17.3 | 18.4 | 18.8 | 18.4 |
| % Mom >34 Years | 6.8 | 6.2 | 5.8 | 5.5 | 5.1 | 4.7 |
| % First Birth | 36.5 | 37.2 | 37.4 | 38.2 | 38.4 | 39.4 |
| % Prior Fetal Death | 10.5 | 10.2 | 10.4 | 10.3 | 10.4 | 10.4 |
| Mean Infant Health Endowment | | | | | | |
| Weight | 3253 | 3264 | 3273 | 3276 | 3285 | 3291 |
| % Very Low Birth Weight | 1.32 | 1.21 | 1.18 | 1.23 | 1.18 | 1.17 |
| % Low Birth Weight | 8.3 | 8.1 | 7.8 | 7.9 | 7.7 | 7.6 |

Notes: Very low birth weight is defined as a birth weight less than 1,500 grams; low birth weight is less than 2,500 grams. The potential sample is limited to the 501 counties with TSPs data in 1970, 1971 and 1972. In any given year, the sample is further restricted to counties with nonmissing TSPs and infant mortality values. All sample means are weighted by the total number of births in the county. See the Data Appendix for information on sources and more details on how the variables were calculated.

Table 2: Differences in County Characteristics by 1971 TSPs Level, 1971-1972 TSPs Change, and 1972 Nonatta

| | Difference in Sample Means between Groups of Counties | | | | |
|---|---|---|---|---|---|
| | High versus Low | Big versus Small | | | Nonattainment versus Att |
| | 1971 TSPs level | 1971-1972 TSPs Reduction | | Unrestricted Sample of Counties | |
| | 1971 level | 1971 level | 1971-72 change | 1971 level | 1971-72 change |
| Internal Infant Mortality Rate | **129.8**** | 21.8 | 18.8 | **132.5**** | -52.6 |
| | (50.6) | (55.5) | (36.7) | (52.7) | (37.8) |
| TSPs Concentration | **45.8**** | **26.3**** | **-25.6**** | **36.5**** | **-11.9**** |
| | (3.6) | (4.9) | (2.0) | (4.5) | (2.4) |
| Total Births [or % Change] | **3539**** | **2481**** | -0.8 | **3973**** | -1.0 |
| | (762) | (767) | (0.6) | (714) | (0.6) |
| County-Level Economic Conditions | | | | | |
| Log Income Per Capita (x 100) | 4.19 | **6.55**** | -0.02 | **6.62*** | **0.67**** |
| | (2.72) | (2.54) | (0.22) | (3.08) | (0.26) |
| Parental Demographic and Socioeconomic Characteristics | | | | | |
| % Mother H.S. Dropout | 0.2 | 3.8 | 0.23 | -0.9 | 0.10 |
| | (2.7) | (2.9) | (0.18) | (2.9) | (0.20) |
| Mother's Years of Educations | -0.07 | -0.09 | **-0.21*** | 0.09 | -0.12 |
| | (0.12) | (0.13) | (0.09) | (0.13) | (0.08) |
| Father's Years of Education | -0.04 | -0.14 | **-0.20*** | 0.15 | -0.13 |
| | (0.14) | (0.14) | (0.10) | (0.15) | (0.10) |
| % Single Mother | 1.45 | -0.24 | 0.24 | 2.77 | 0.37 |
| | (1.46) | (1.62) | (0.24) | (1.47) | (0.22) |
| % Black | **6.6**** | 1.6 | 0.23 | **5.7*** | 0.30 |
| | (2.2) | (2.7) | (0.20) | (2.5) | (0.17) |
| % Foreign-born | **3.5*** | **5.5**** | | -1.1 | |
| | (1.7) | (1.7) | | (2.8) | |
| Medical Services Utilization | | | | | |
| % No Prenatal Care | **0.62**** | **0.60**** | 0.06 | -0.08 | 0.02 |
| | (0.21) | (0.22) | (0.06) | (0.37) | (0.07) |
| % Prenatal Care in 1st Trimester | -3.1 | 4.9 | -0.19 | 6.5 | 0.21 |
| | (3.6) | (3.9) | (0.43) | (4.0) | (0.44) |
| Maternal Health Endowment | | | | | |
| % Teenage Mother | **1.43*** | -0.99 | 0.26 | 0.96 | -0.02 |
| | (0.70) | (0.69) | (0.15) | (0.82) | (0.16) |
| % Mom>34 Years-old | 0.19 | **0.55**** | -0.04 | 0.06 | 0.004 |
| | (0.20) | (0.18) | (0.07) | (0.21) | (0.08) |
| % First Birth | 0.29 | **-1.36*** | 0.20 | 0.55 | 0.39 |
| | (0.66) | (0.61) | (0.43) | (0.93) | (0.40) |
| % Prior Fetal Death | -1.15 | -0.40 | 0.30 | -0.57 | 0.05 |
| | (0.92) | (0.99) | (0.17) | (0.90) | (0.19) |
| Number of Counties | 501 | 501 | 501 | 501 | 501 |

Notes: See notes to Table 1. Entries are the differences in the weighted means of the variables (where the weight is the total number of births) ac standard errors of the differences are in parentheses and allow for unequal variances between county groups. The first column contains the mean between counties with 1971 TSPs concentrations above and below the median 1971 county-level TSPs concentration. The next two columns cor levels and 1971-72 changes between counties with a 1971-72 change in TSPs that is below and above the median change in TSPs. The final fou and 1971-72 changes between nonattainment and attainment counties. The sample consists of the 501 counties with non-missing TSPs data in 1 final two columns where the sample is further restricted to the 176 counties with 1970 geometric mean TSPs concentrations between 60 and 90 n * indicates significance at 5% level, ** indicates significance at 1% level

Table 3: Cross-Sectional Estimates of the Association between Mean TSPs and Infant Mortality Rates
(estimated standard errors in parentheses)

| | Infant Deaths Due to Internal Causes (per 100,000 Live Births) | | | | | |
|---|---|---|---|---|---|---|
| | (1) | (2) | (3) | (4) | (5) | (6) |
| 1969 Cross-Section | **2.48** | 0.63 | 0.22 | -0.14 | 0.38 | 0.20 |
| | **(0.92)** | (0.40) | (0.39) | (0.38) | (0.42) | (0.41) |
| | [412,.05] | [406,.55] | [403,.65] | [357,.69] | [406,.69] | [357,.75] |
| 1970 Cross-Section | 1.30 | **0.80** | 0.52 | 0.26 | 0.42 | -0.07 |
| | (0.72) | **(0.41)** | (0.33) | (0.28) | (0.34) | (0.24) |
| | [501,.02] | [490,.47] | [482,.55] | [441,.60] | [490,.57] | [441,.67] |
| 1971 Cross-Section | 1.59 | 0.88 | 0.25 | -0.05 | **1.38** | 0.75 |
| | (0.98) | (0.51) | (0.48) | (0.44) | **(0.47)** | (0.47) |
| | [501,.02] | [495,.48] | [482,.57] | [460,.62] | [495,.60] | [460,.68] |
| 1972 Cross-Section | 0.89 | -0.30 | -0.83 | **-1.32** | -0.94 | **-1.82** |
| | (1.20) | (0.84) | (0.74) | **(0.65)** | (0.82) | **(0.87)** |
| | [501,.00] | [492,.33] | [483,.43] | [455,.48] | [492,.46] | [455,.57] |
| 1973 Cross-Section | 2.51 | 1.27 | -0.13 | -1.06 | **2.79** | 0.41 |
| | (1.52) | (0.84) | (0.77) | (0.79) | **(0.70)** | (0.81) |
| | [495,.02] | [488,.46] | [477,.57] | [454,.59] | [488,.58] | [454,.66] |
| 1974 Cross-Section | **2.88** | **2.01** | **1.41** | 1.01 | **3.17** | **2.04** |
| | **(1.34)** | **(0.81)** | **(0.60)** | (0.67) | **(0.73)** | **(0.80)** |
| | [489,.03] | [484,.45] | [475,.57] | [455,.61] | [484,.60] | [455,.68] |
| 1969-1974 Pooled | **2.54** | **1.69** | 0.42 | 0.16 | **1.84** | 0.22 |
| | **(0.84)** | **(0.48)** | (0.28) | (0.22) | **(0.45)** | (0.20) |
| | [2899,.04] | [2855,.40] | [2802,.54] | [2622,.58] | [2855,.46] | [2622,.61] |
| Basic Natality Vars. | N | Y | Y | Y | Y | Y |
| Unrestricted Natality | N | N | Y | Y | N | Y |
| Income, Employment | N | N | N | Y | N | Y |
| Income Assist. Sources | N | N | N | Y | N | Y |
| State Effects | N | N | N | N | Y | Y |

Notes: See notes to Table 1. Numbers in brackets are the number of counties and R-squareds of the regressions, respectively. The potential sample is limited to the 501 counties with TSPs data in 1970, 1971 and 1972. In a given year, the sample is further restricted to counties with nonmissing covariates. Sampling errors are estimated using the Eicker-White formula to correct for heteroskedasticity. The sampling errors in the "1969-1974 Pooled" row are also corrected for county-level clustering in the residuals over time. Regressions are weighted by numbers of births in each county. Internal causes of death arise from common health problems, such as respiratory and cardiopulmonary deaths. The control variables are listed in the Data Appendix and in Table 1. State Effects are separate indicator variables for each state. Bold text indicates that the null hypothesis that the estimate is equal to zero can be rejected at the 5% level.

Table 4: Fixed Effects Estimates of Association between Mean TSPs and Infant Mortality Rates
(estimated standard errors in parentheses)

| | Infant Deaths Due to Internal Causes (per 100,000 Live Births) | | | | | |
|---|---|---|---|---|---|---|
| | (1) | (2) | (3) | (4) | (5) | (6) |
| 1969-1974 Pooled | | | | | | |
| Mean TSPs | **1.83** | 0.62 | -0.23 | -0.28 | -0.27 | -0.18 |
| | **(0.54)** | (0.37) | (0.26) | (0.26) | (0.27) | (0.27) |
| R-squared | 0.62 | 0.67 | 0.73 | 0.73 | 0.75 | 0.77 |
| Dep. Var. Mean | 1795 | 1794 | 1794 | 1797 | 1794 | 1797 |
| Sample Size | 2899 | 2855 | 2802 | 2622 | 2855 | 2622 |
| 1971-1972 First Differenced | | | | | | |
| Mean TSPs | 0.74 | 0.47 | -0.14 | -0.31 | -0.69 | -0.53 |
| | (0.71) | (0.71) | (0.67) | (0.67) | (0.75) | (0.80) |
| R-squared | 0.00 | 0.05 | 0.13 | 0.17 | 0.18 | 0.30 |
| Dep. Var. Mean | -54.6 | -53.7 | -51.2 | -49.7 | -53.7 | -49.7 |
| Sample Size | 501 | 489 | 474 | 449 | 489 | 449 |
| County Fixed Effects | Y | Y | Y | Y | Y | Y |
| Basic Natality Vars. | N | Y | Y | Y | Y | Y |
| Unrestricted Natality | N | N | Y | Y | N | Y |
| Income, Employment | N | N | N | Y | N | Y |
| Income Assist. Sources | N | N | N | Y | N | Y |
| Year Effects | N | N | Y | Y | Y | Y |
| State-Year Effects | N | N | N | N | Y | Y |

Notes: In the first panel the regressions are based on pooled data and include county indicator variables as controls. In the second panel the regressions are based on first-differenced data. Standard errors are estimated using the Eicker-White formula to correct for heteroskedasticity. Regressions are weighted by numbers of births in each county and year in the first panel and the sum of total births in 1971 and 1972 in the second panel. Bold text indicates that the null hypothesis that the estimate is equal to zero can be rejected at the 5% level.

Table 5: Reduced-Form Estimates of the Impact of 1972 Nonattainment Status on 1971-1972 Changes in TSPs Pollution and Internal Infant Mortality Rates
(estimated standard errors in parentheses)

| | (1) | (2) | (3) | (4) | (5) | (6) |
|---|---|---|---|---|---|---|
| 1971-72 Change in Mean TSPs | | | | | | |
| TSPs Nonattain. in 1972 | -11.9 | -11.0 | -10.0 | -8.8 | -8.9 | -6.3 |
| | (2.4) | (2.4) | (2.3) | (2.2) | (2.0) | (2.0) |
| F-stat. Nonattainment | 23.5 | 21.5 | 19.5 | 15.3 | 20.5 | 9.6 |
| (numerator d.o.f.) | (1) | (1) | (1) | (1) | (1) | (1) |
| F-stat. Other variables | ----- | 0.9 | 1.4 | 1.6 | 1.4 | 1.6 |
| (numerator d.o.f.) | ----- | (11) | (43) | (54) | (11) | (54) |
| F-stat. State-Year Effects | ----- | ----- | ----- | ----- | 17.3 | 3.5 |
| (numerator d.o.f.) | ----- | ----- | ----- | ----- | (49) | (49) |
| R-Squared | 0.06 | 0.08 | 0.17 | 0.19 | 0.30 | 0.40 |
| Dep. Variable Mean | -11.3 | -11.3 | -11.3 | -11.1 | -11.3 | -11.1 |
| 1971-72 Change in Infant Mortality | | | | | | |
| TSPs Nonattain. in 1972 | -52.6 | -78.6 | -84.6 | -68.3 | -115.6 | -95.1 |
| | (37.8) | (38.9) | (38.3) | (39.1) | (42.5) | (46.1) |
| F-stat. Nonattainment | 1.9 | 4.1 | 4.9 | 3.1 | 7.4 | 4.3 |
| (numerator d.o.f.) | (1) | (1) | (1) | (1) | (1) | (1) |
| F-stat. Other variables | ----- | 1.6 | 1.9 | 2.1 | 1.9 | 2.1 |
| (numerator d.o.f.) | ----- | (11) | (43) | (54) | (11) | (54) |
| F-stat. State-Year Effects | ----- | ----- | ----- | ----- | 6.6 | 3.8 |
| (numerator d.o.f.) | ----- | ----- | ----- | ----- | (49) | (49) |
| R-Squared | 0.00 | 0.05 | 0.14 | 0.17 | 0.18 | 0.31 |
| Dep. Variable Mean | -54.6 | -53.7 | -51.2 | -49.7 | -53.7 | -49.7 |
| Basic Natality Vars. | N | Y | Y | Y | Y | Y |
| Unrestricted Natality | N | N | Y | Y | N | Y |
| Income, Employment | N | N | N | Y | N | Y |
| Income Assist. Sources | N | N | N | Y | N | Y |
| State-Year Effects | N | N | N | N | Y | Y |
| Sample Size | 501 | 489 | 474 | 449 | 489 | 449 |

Notes: The dependent variables are the 1971-72 first-differences of mean TSPs pollution and 1971-72 first-differences in infant deaths due to internal causes per 100,000 live births. Nonattainment in 1972 is an indicator variable equal to one if the county had TSPs concentrations above the federal air quality standard for TSPs in 1970. Standard errors are estimated using the Eicker-White formula to correct for heteroskedasticity. Regressions are weighted by numbers of births in each county and year.

Table 6: Instrumental Variables Estimates of the Effect of Mean TSPs on Infant Mortality Rates, Based on 1971-72 Changes Using 1972 Nonattainment Status as Instrument (estimated standard errors in parentheses)

| | 1971-1972 Change in Infant Deaths (per 100,000 Live Births) | | | | | |
|---|---|---|---|---|---|---|
| | (1) | (2) | (3) | (4) | (5) | (6) |
| Change in Mean TSPs | 4.43 | 7.14 | 8.48 | 7.78 | 13.06 | 15.08 |
| | (3.14) | (3.65) | (4.12) | (4.80) | (5.63) | (8.71) |
| Basic Natality Vars. | N | Y | Y | Y | Y | Y |
| Unrestricted Natality | N | N | Y | Y | N | Y |
| Income, Employment | N | N | N | Y | N | Y |
| Income Assist. Sources | N | N | N | Y | N | Y |
| State-Year Effects | N | N | N | N | Y | Y |
| Sample Size | 501 | 489 | 474 | 449 | 489 | 449 |

Notes: Results are from two-stage least squares estimation using 1971-72 first-differences, with changes in mean TSPs instrumented by nonattainment status in 1972. Estimated standard errors allow for heteroskedasticity. Regressions are weighted by numbers of births in each county and year.

Table 7: Instrumental Variables Estimates for Infant Deaths within 1-Year, 1-Month, and 1-Day of Birth
For Counties with 1970 Geometric Mean TSPs near Regulatory Threshold
(estimated standard errors in parentheses)

| | 1971-72 Change in Infant Deaths Due to Internal Causes (per 100,000 Live Births) | | | | | |
|---|---|---|---|---|---|---|
| | | | 1970 Geometric Mean TSPs in the Range of | | | |
| | All Counties | | 50-115 $\mu g/m^3$ | | 60-90 $\mu g/m^3$ | |
| | (1) | (2) | (1) | (2) | (1) | (2) |
| Deaths w/in 1 year | | | | | | |
| Mean TSPs | 7.14 | 13.06 | 11.32 | 11.59 | 15.22 | 17.37 |
| | (3.65) | (5.63) | (8.72) | (6.84) | (8.94) | (11.56) |
| Deaths w/in 28 days | | | | | | |
| Mean TSPs | 4.23 | 6.57 | 8.15 | 7.41 | 12.48 | 12.60 |
| | (2.91) | (4.38) | (6.88) | (5.60) | (7.07) | (9.39) |
| | [59.2] | [50.3] | [72.0] | [63.9] | [82.0] | [72.5] |
| Deaths w/in 24 hours | | | | | | |
| Mean TSPs | 1.69 | 2.37 | 1.12 | 1.60 | 3.40 | 3.20 |
| | (2.19) | (3.14) | (4.93) | (4.07) | (4.74) | (7.01) |
| | [23.7] | [18.1] | [9.9] | [13.8] | [22.3] | [18.4] |
| Basic Natality Vars. | Y | Y | Y | Y | Y | Y |
| State-Year Effects | N | Y | N | Y | N | Y |
| Sample Size | 489 | 489 | 320 | 320 | 173 | 173 |

Notes: The dependent variables are the 1971-72 first-differences in the number of infant deaths due to internal causes within one-year, 28-days, and 24-hours of birth (per 100,000 live births). The columns correspond to subsamples of counties with annual geometric mean readings of TSPs in 1970 in the specified range. Results are from two-stage least squares estimation, with 1971-72 changes in mean TSPs instrumented by nonattainment status in 1972. Estimated standard errors allow for heteroskedasticity and are in parentheses. In the second and third panels, the ratio of that panel's estimated coefficient to the first panel's coefficient is reported in brackets. Regressions are weighted by numbers of births in each county and year.

Table 8: Internal Validity of the Instrumental Variables Estimates of the Effect of Mean TSPs on Infant Mortality, Based on 1971-1972 Changes
(estimated standard errors in parentheses)

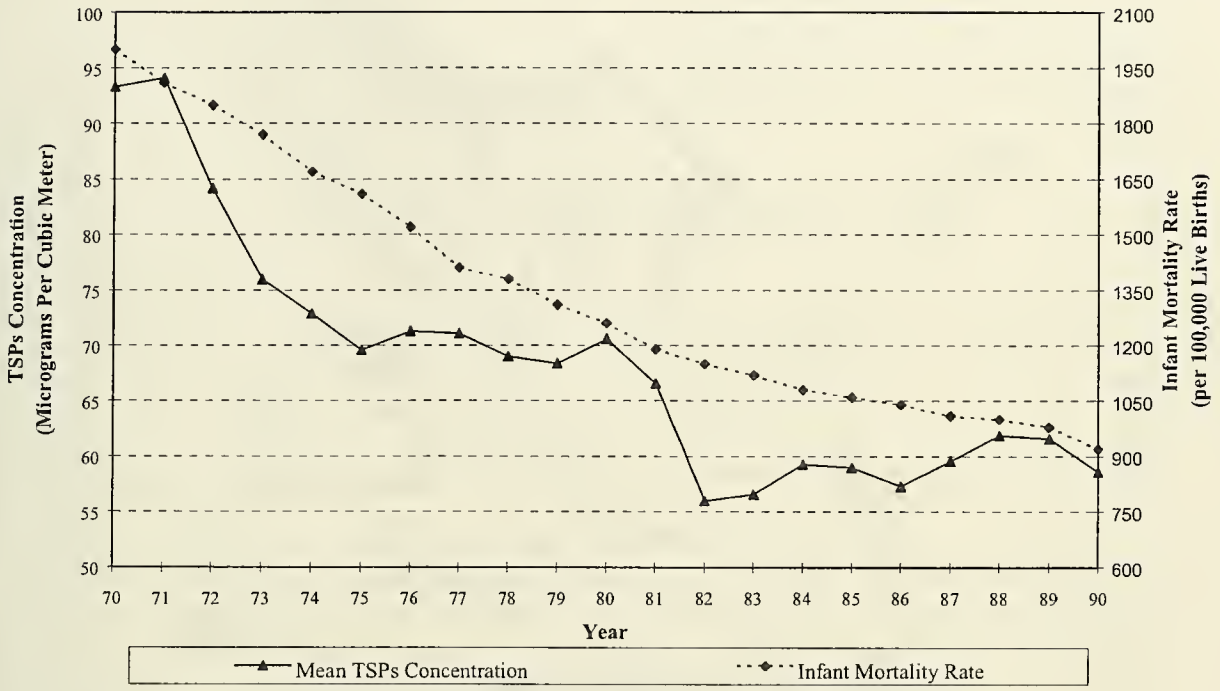
| | 1971-72 Change in Infant Deaths (per 100,000 Live Births) | | | | | |
|---|---|---|---|---|---|---|
| | | | 1970 Geometric Mean TSPs in the Range of | | | |
| | All Counties | | 50-115 $\mu g/m^3$ | | 60-90 $\mu g/m^3$ | |
| | (1) | (2) | (1) | (2) | (1) | (2) |
| External Deaths | | | | | | |
| Mean TSPs | -0.92 | -0.95 | -2.06 | -0.75 | -2.16 | -1.90 |
| | (0.74) | (1.09) | (1.87) | (1.39) | (1.78) | (2.08) |
| Sample Size | 489 | 489 | 320 | 320 | 173 | 173 |
| Control for Change in "Premature Nurseries" | | | | | | |
| Mean TSPs | 7.33 | 13.43 | 11.69 | 10.88 | 16.11 | 17.25 |
| | (3.95) | (5.89) | (9.41) | (6.56) | (9.97) | (11.56) |
| Sample Size | 487 | 487 | 319 | 319 | 172 | 172 |
| Controls for Quadratic in 1970 TSPs levels | | | | | | |
| Mean TSPs | 8.32 | 11.41 | 20.51 | 35.48 | 17.16 | 26.37 |
| | (7.34) | (8.08) | (13.20) | (28.18) | (14.19) | (20.42) |
| Sample Size | 488 | 488 | 320 | 320 | 173 | 173 |
| Basic Natality Vars. | Y | Y | Y | Y | Y | Y |
| State-Year Effects | N | Y | N | Y | N | Y |

Notes: In the first panel, the dependent variable is the 1971-72 first-differences in number of infant deaths due to external causes, such as accidents and homicides, within one-year of birth (per 100,000 live births). In the second panel, each specification includes a control for the change in the county-level number of "premature nurseries." In the third panel, each specification controls for the geometric mean of TSPs in 1970 and its square. The columns correspond to subsamples of counties with annual geometric mean readings of TSPs in 1970 in the specified range. Results are from two-stage least squares estimation, with 1971-72 changes in mean TSPs instrumented by nonattainment status in 1972. Estimated standard errors allow for heteroskedasticity. Regressions are weighted by numbers of births in each county and year.

Appendix Table 1
Key Dates Associated with the Clean Air Act Amendments of 1970

| Date | Event |
|---|---|
| December 31, 1970 | President Nixon Signs 1970 Clean Air Act Amen |
| April 30, 1971 | EPA Sets National Ambient Air Quality Standard Must be Achieved by 1975, with possibility of a 2 Extension |
| August 14, 1971 | EPA Publishes Requirements for Preparation, Ad and Submittal of State Implementation Plans in *C Federal Regulations* |
| January, 1972 | All State Implementation Plans Due to EPA |

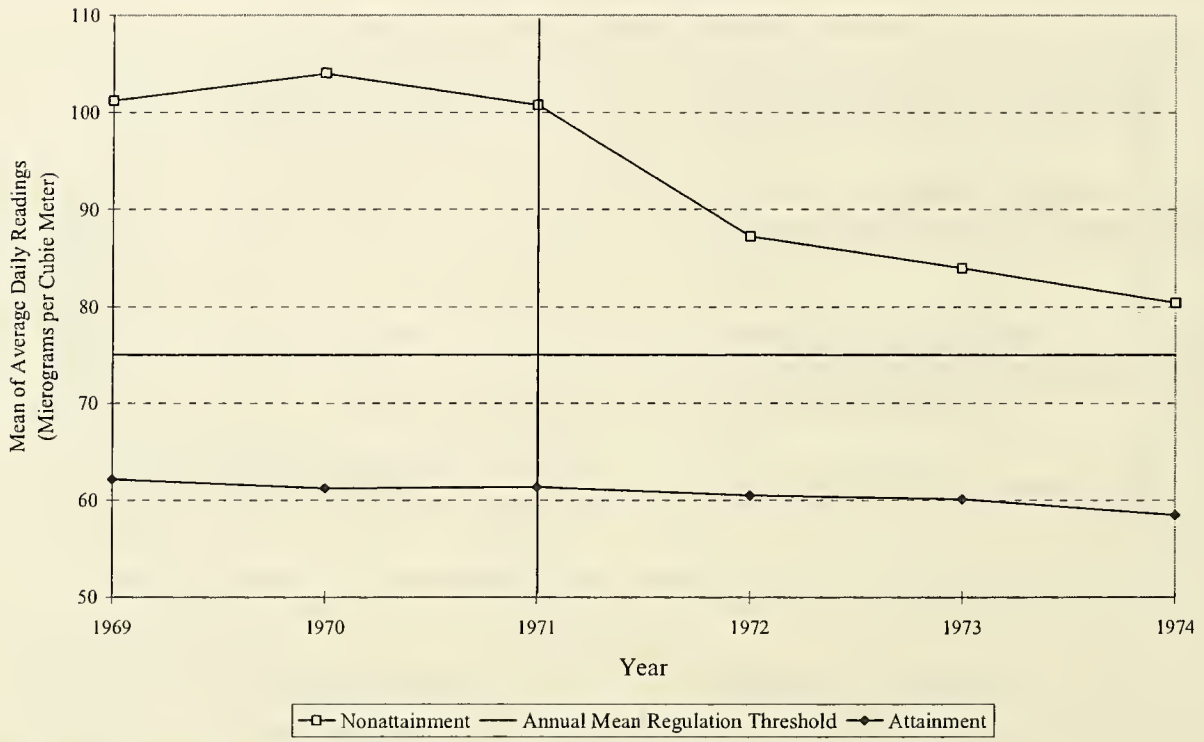
Figure 1: National Trends in Total Suspended Particulates Air Pollution and Infant Mortality Rates




Source: Authors' tabulations from EPA's "Quick Look Reports" data file and U.S. National Center for Health Statistics.

Figure 2: Trends in TSPs Pollution and Infant Mortality, by 1972 Nonattainment Status

A. Trends in Mean TSPs Concentrations, by 1972 Nonattainment Status




Source: Authors' tabulations from EPA's "Quick Look Reports" data file.

B. Trends in Internal Infant Mortality Rate, by 1972 Nonattainment Status

Figure 3: Nonattainment Status for TSPs Pollution in 1972, by County

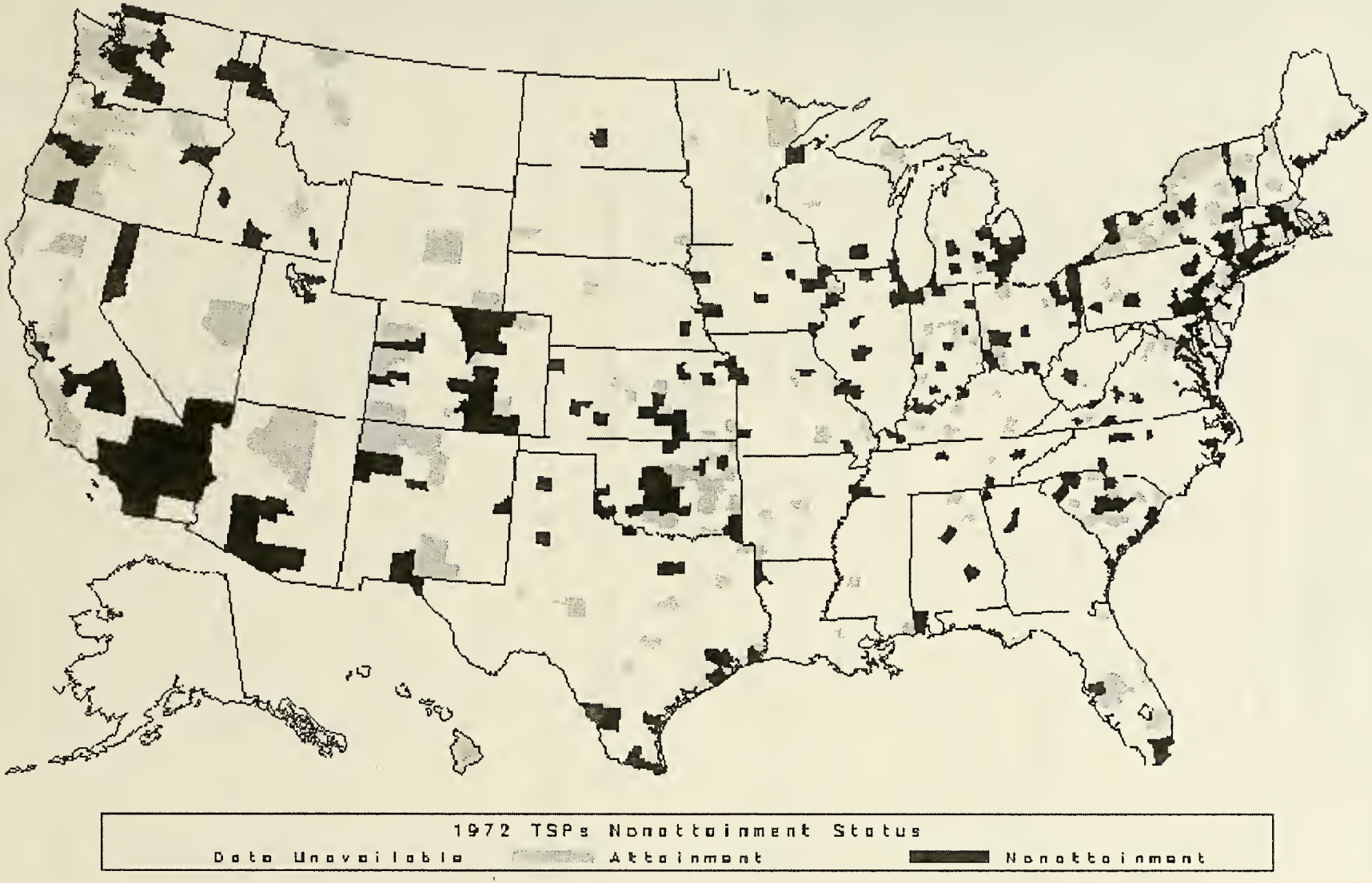
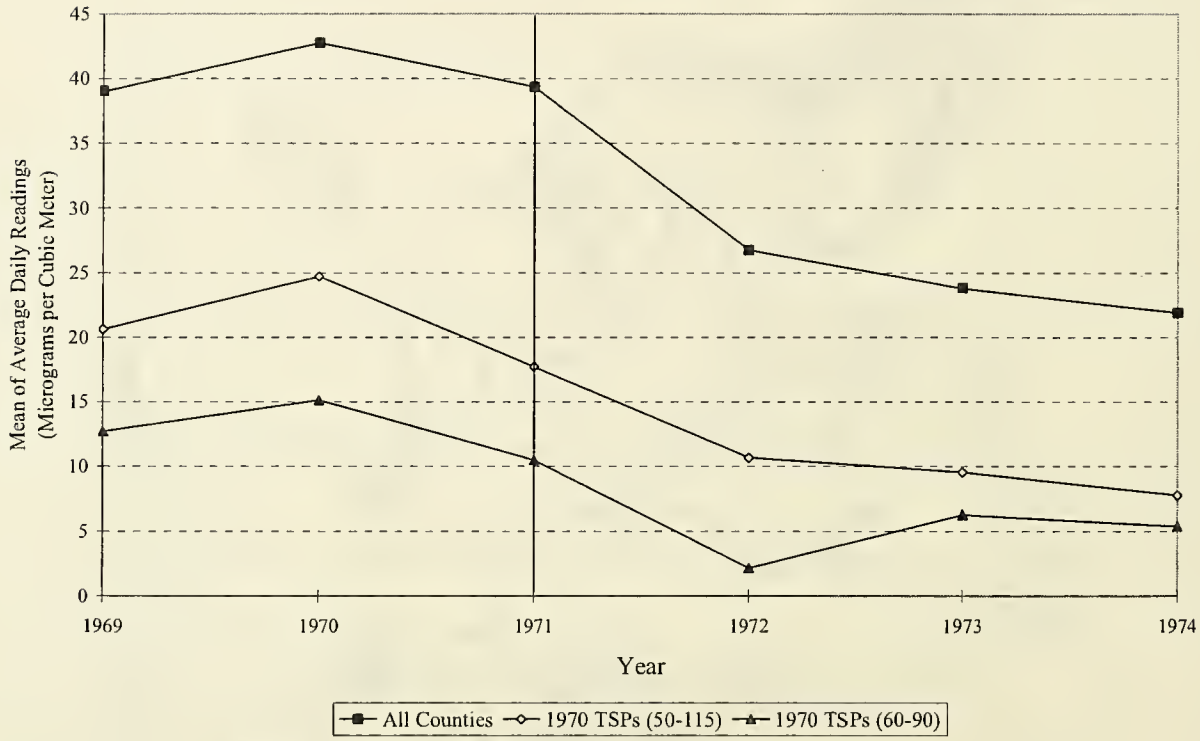


Figure 4: Raw Nonattainment-Attainment Differences in TSPs and Infant Mortality, By 1970 TSPs Ranges

A. Nonattainment-Attainment TSPs Differences by 1970 TSPs Ranges




B. Nonattainment-Attainment Mortalilty Differences by 1970 TSPs Ranges

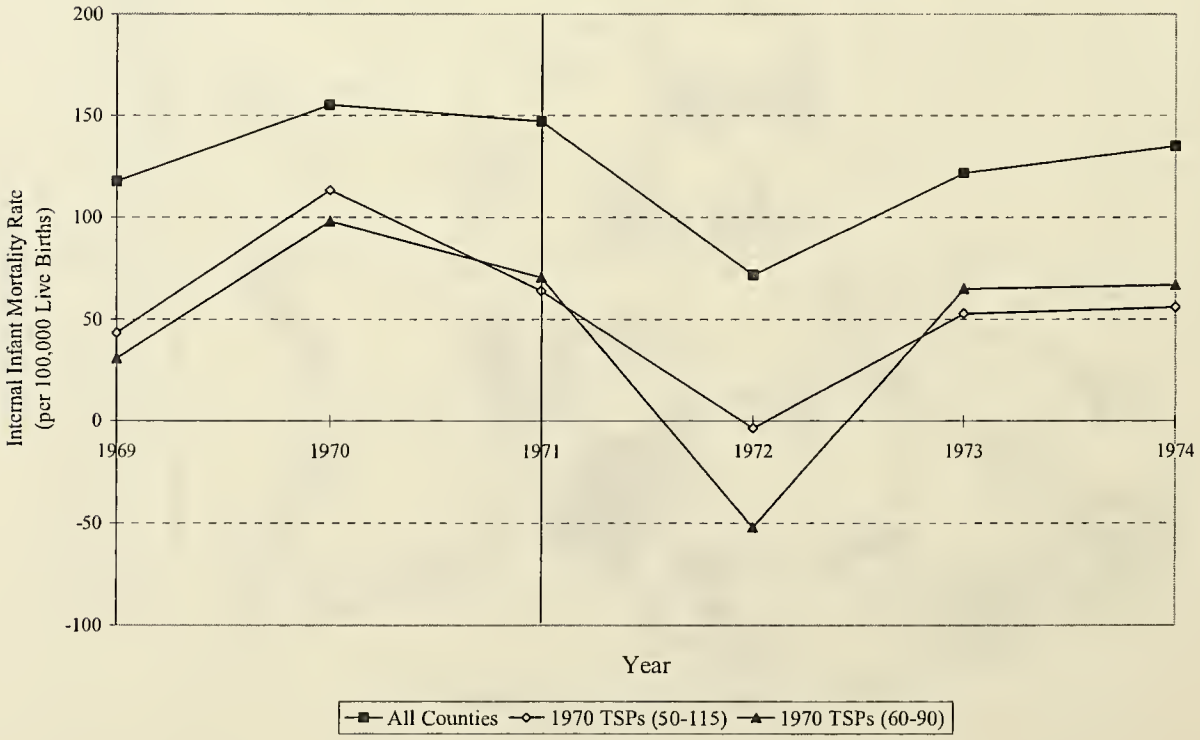
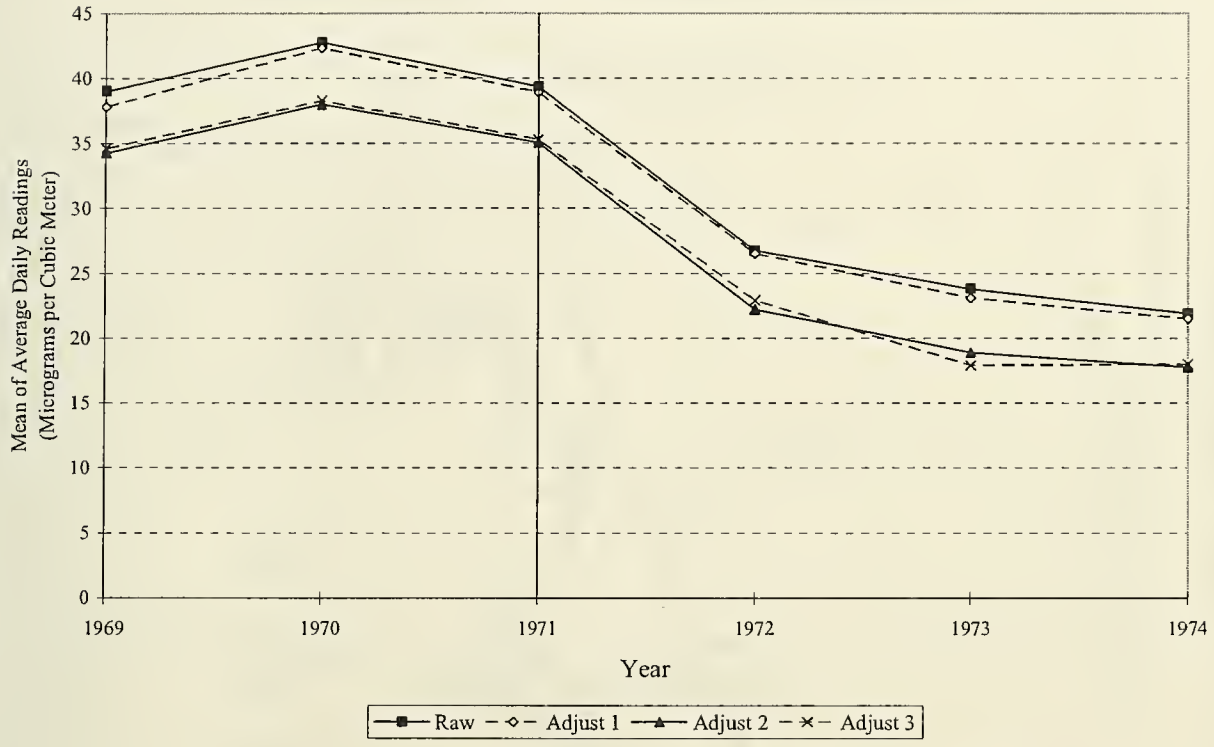


Figure 5: Raw and Regression-Adjusted Nonattainment-Attainment Differences in TSPs and Infant Mortality, All Counties 1969-1974

A. Nonattainment-Attainment TSPs Differences, Raw and Adjusted




B. Nonattainment-Attainment Mortality Differences, Raw and Adjusted

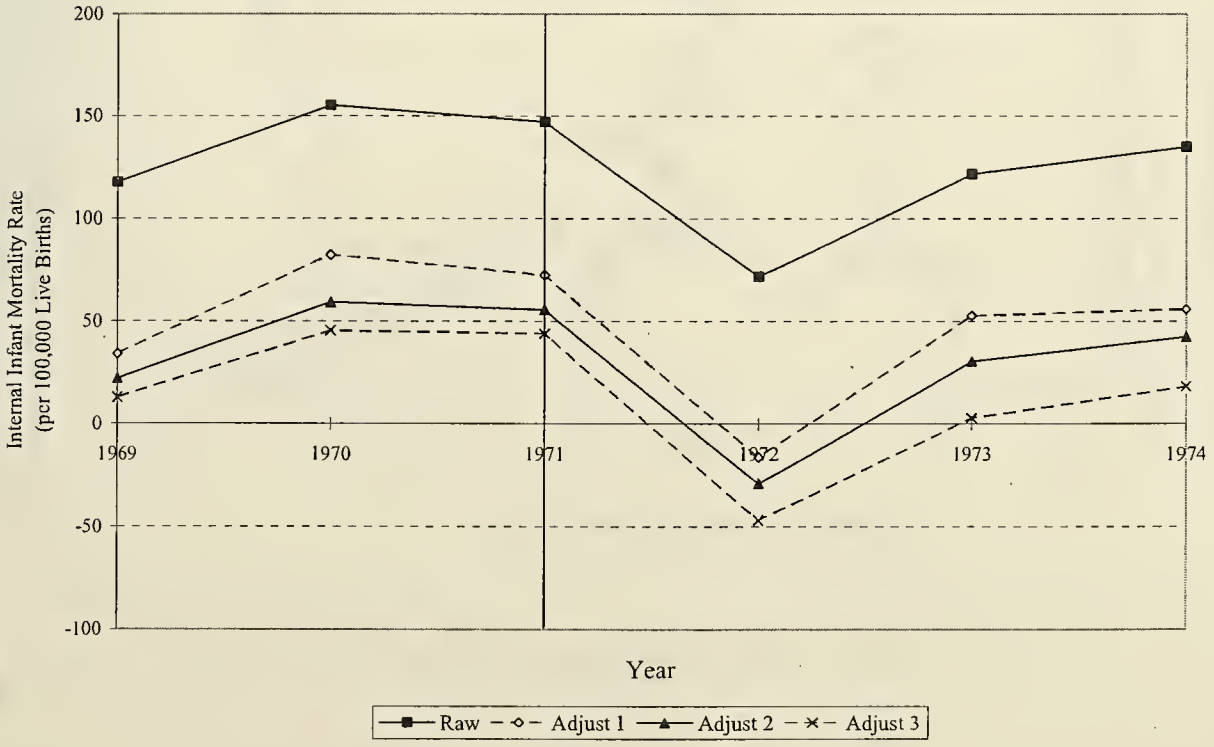


Figure 6: 1971-72 Post-Regulation Changes in Mean TSPs and Internal Infant Mortality Rates
By Geometric Mean TSPs in Regulation Selection Year, 1970

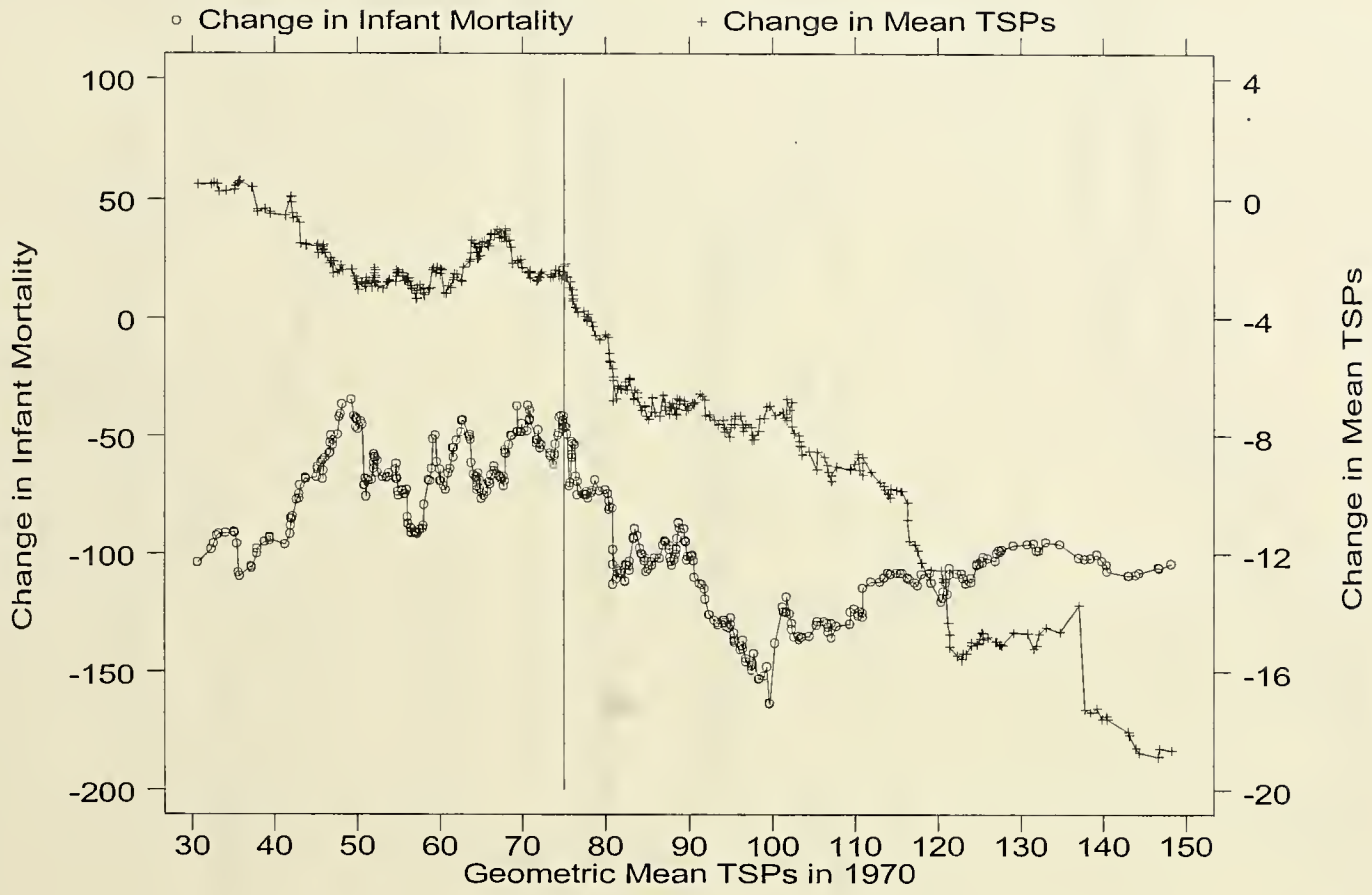
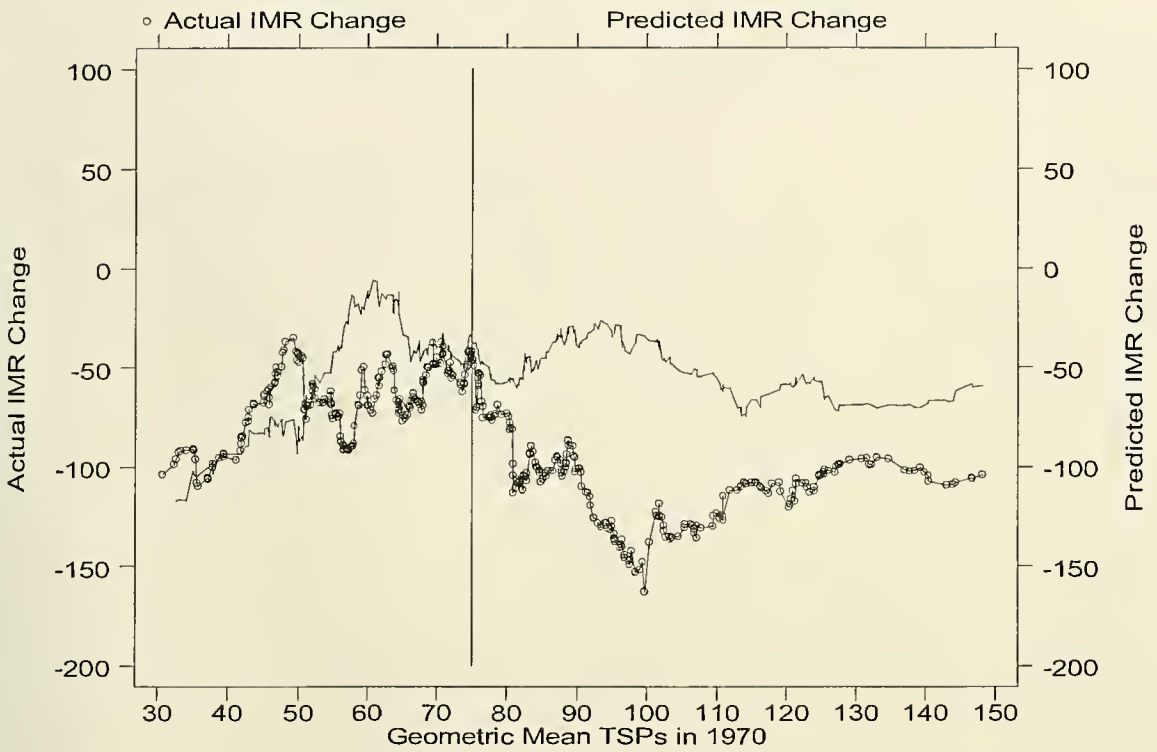
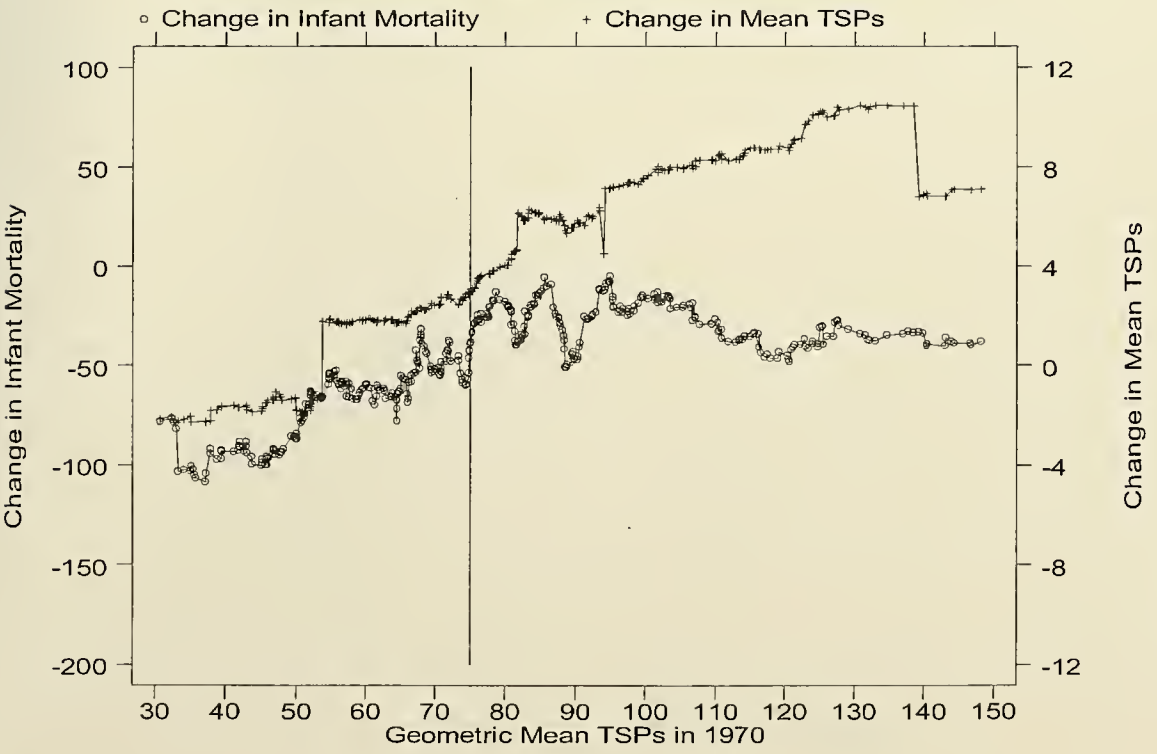


Figure 7: Validity Checks of the Effects of 1972 TSPs Nonattainment Status

A. 1971-72 Post-Regulation Changes in Actual and Predicted Infant Mortality Rates




B. 1969-70 Pre-Regulation Changes in Mean TSPs and Infant Mortality Rates



Appendix Figure 1: Predicted Nonattainment-Attainment Mortality Differences Based on the Variables in the Adjust 3 Regression Specification Excluding TSPs